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ASSOCIATION BETWEEN GLYCATED HEMOGLOBIN (HbA1c) AND LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disease arising from a complex genetic, behavioral, and environmental relationship. Morbidity and mortality arising from DM are strongly attributed to lifestyle changes such as an unhealthy diet, body weight, physical inactivity, obesity, and tobacco use.[1] T2DM is an increasingly growing public health concern worldwide with a major impact on the country's wellbeing, quality of life, and health care system. Diabetes mellitus is characterized by chronic hyperglycemia with carbohydrate, protein, and fat metabolism issues caused by both insulin secretion and insulin action deficiencies. Glucose and lipid metabolism are interrelated. Dyslipidemia in diabetic patients is well known to be considered the most risk factor for macrovascular complications.[2,3]

Diabetes mellitus is a metabolic disorder that affects multiple body systems in the long term. One category within the continuum of diseases is cardiovascular disease (CVD); impaired lipid metabolism in atherosclerosis requires a diabetes mellitus.[4] Studies show that 12.1% of urban Indians are diabetic, with onset of symptoms occurring around decade before in the West.[5] Cardiovascular disease (CVD) is the primary cause of death in people with diabetes mellitus (DM), and diabetes is a major risk factor for CVD. In addition to the kidney and eye problems known as microvascular consequences of diabetes, macrovascular diseases involving the heart, blood vessels, and other vital organs are on the rise as well.[6]

As a worldwide epidemic, obesity-related T2D has serious health consequences. Increased morbidity and mortality from cardiovascular disease is seen in patients with type 2 diabetes and obesity.[7] Enhanced glycemic control can be demonstrated with the help of the A1C test, which is recommended for use in clinical trials. It's the gold standard for keeping tabs on your blood sugar levels. For persons with diabetes who use insulin, keeping track of their blood sugar levels on their own can be a big help in keeping their health and medication regimen in check. Patients with Type II diabetes (T2DM) are at an increased risk of cardiovascular disease due to the prevalence of dyslipidemia in this population (CVDs). Further more to a valid glycemic index, glycated haemoglobin may be used as a predictor of dyslipidemia.[8]

Consequently, HbA1c values can be used as a marker of lipid profile imbalance, making them a useful tool in the prevention of cardiovascular complications in people with type II diabetes

mellitus.[9] This dramatically raises CVD risk as opposed to people without DM. CVD distribution in people with T2DM is higher and is also the major killer in this population.[10] The role of hyperglycemia in Cardiovascular Disease (CVD) is supported by a direct correlation between fasting blood glucose and cardiovascular events.[3] Uncontrolled hyperglycemia has been linked to impaired lipid metabolism, which in turn has been linked to cardiovascular problems in people with diabetes. Plasma lipid and lipoprotein abnormalities have been linked to type 2 diabetes, according to published investigations. Hyperglycemia has been linked directly to coronary artery disease in a number of epidemiological and clinical investigations.[11]

Even while hypertriglyceridemia is more common in non-insulin-dependent diabetes mellitus, it is also linked to insulin resistance. Hypertriglyceridemia is a hallmark of insulin resistance. If insulin resistance exists, sugar cannot enter cells. The pancreas responds by producing even more insulin, leading to a systemic surplus. Hypertriglyceridemia and insulin resistance may be related to a competition between glucose and lipid substrates, but this is not well understood.[3,12]

In developing countries, the prevalence of T2DM increases with age, most people with diabetes are between 45 and 64 years of age, while in developed countries the majority of patients with diabetes are 65 years of age and older.[13] Lately, a metabolic syndrome associated with obesity has gained widespread attention. T2DM and the body mass index (BMI) have been used as a composite covariate for adjustment in Type 2 diabetes mellitus studies to compensate for the association between obesity. A direct relation between increasing BMI and increasing TC, LDL-C, and TG and an inverse correlated with HDL-C has emerged in some Studies. This association between BMI and the degree of lipoprotein in particular LDL-C has been suggested as a strong participating risk factor for cardiovascular diseases in obese people.[14]

Glycated hemoglobin (HbA1c) is a symbol used for long-term control of glycemia. It also predicts the risk of developing diabetic complications in diabetic patients, in addition to its role as a signal for a blood glucose level. In diabetics, levels of hemoglobin A1c (HbA1c) are regularly measured to assess their glycemic regulation. The goal is to reach a grade below 7%. Several factors can affect the degree of HbA1c, including exercise, sugar intake, and medication adherence. In some studies, it was stated that HbA1c could potentially be used for predicting cardiovascular disease (CVD) and dyslipidemia as a possible biomarker.[15,16] Glycated

hemoglobin (HbA1c) levels are routinely measured in diabetics to monitor their glycemic control. The goal is to achieve a level below 7%. Levels of HbA1c can be affected by multiple factors, including sugar intake, exercise and adherence to medications. Some studies have reported that HbA1c could potentially be utilized as a possible biomarker for predicting dyslipidemia and cardiovascular disease.[15,17]

The chance of developing coronary heart disease is greatly increased by dyslipidemia. In spite of the fact that DM and dyslipidemia are two different risk factors for atherosclerosis, many research on individuals with type 2 DM have shown that DM and dyslipidemia have a wide variety of pathophysiological overlaps and that these interactions speed up the process of atherogenesis.[18] HbA1c has been proposed as a biomarker for dyslipidemia and glycemic management in patients with type 2 diabetes mellitus, according to several research.[19] HbA1c predicts the risk of developing diabetic complications in patients with DM. Apart from classical risk factors like dyslipidemia, elevated HbA1c is an independent risk factor for CVD. Many studies have proposed HbA1c to be used as a biomarker of both glycemic control and dyslipidemia in type 2 diabetes mellitus.[19]

In addition, diabetes mellitus is considered a metabolic disorder, and hyperlipidemia is connected with it. The prevalence of this condition differs based on the type and severity of diabetes, the person's glycemic regulation, their nutritional state, their age, and other factors.[20] Plasma lipid and lipoprotein abnormalities, such as decreased HDL cholesterol, small dense LDL particle predominance, and high triglycerides, are all associated with type 2 diabetes. These plasma lipid and lipoprotein abnormalities are interrelated and form a cluster.[21]

The purpose of this study was to investigate whether or not patients with type 2 diabetes mellitus have a correlation between their glycated haemoglobin (HbA1c) levels and their lipid profiles.

AIM AND OBJECTIVES

Aim:

The aim of this study is to assess the relationship between glycemic control (assessed by measuring HbA1c levels) and serum lipid profile as well as to evaluate the importance of HbA1c as an indicator of dyslipidemia in patients with T2DM.

Objectives:

The aim of the study would be achieved with the help of following objectives:

- To determine the relation of HbA1c with Total Cholesterol.
- To determine the relation of HbA1c with Serum Triglyceride.
- To determine the relation of HbA1c with HDL.
- To determine the relation of HbA1c with LDL.
- To determine the relation of HbA1c with VLDL.

REVIEW OF LITERATURE

Diabetes

In about 1500 B.C., the Egyptians recognised diabetes as a rare disease marked by frequent urination and weight loss. The term diabetes mellitus is derived from the Greek word diabetes, which means to siphon or pass through, and the Latin word mellitus, which means sweet. However, it wasn't until 1776 that Matthew Dobson found that the glucose content in the urine of such persons was high.[22] Prior to the beginning of the 18th century, its origin and treatment were unknown, and it was thought to be fatal in every instance. While knowledge of the illness has risen with each passing decade, so too has the disease's effect. In the last century, lifestyle changes and globalization have had a significant influence on civilizations, political institutions, the environment, and human behavior. In both industrialized and developing countries, the incidence of diabetes and obesity has grown rapidly. Diabetes has been termed "the diabetic apocalypse" because it is a worldwide pandemic and a danger to human health and the global economy.[23] Insulin deficiency has developed into a combination of insulin resistance and reduced insulin production in obese individuals.

Epidemiology

Diabetes is among the top 10 major causes of death in this century, along with cardiovascular disease (CVD), respiratory illness, and cancer. It is one of the most significant threats to world health that we have faced in this century.[24,25] According to the World Health Organization (WHO), noncommunicable diseases (NCDs) were responsible for 74% of deaths worldwide in 2019. Diabetes was responsible for 1.6 million deaths, making it the ninth leading cause of death worldwide. Other NCDs include cardiovascular disease, cancer, and Alzheimer's disease. The International Diabetes Federation (IDF) estimates that diabetes affects 8.8% of the adult population, with males having a slightly greater prevalence of the disease than women (9.6% vs 9.0%).[25] According to the most recent figures available for the whole world, 463 million people have diabetes, while 374 million people have impaired glucose tolerance (IGT), which is a disease that may lead to diabetes. According to the International Diabetes Federation (2019), it is anticipated that by the year 2045, the number of people living with diabetes would have

increased by 51%, reaching 700 million, while the number of individuals living with IGT will have increased to 548 million.[26]

Type 2 diabetes, which accounts for 90% of all cases of diabetes, was once thought of as a disease primarily affecting wealthy countries in the "Western" world. However, in recent years, it has become a worldwide epidemic that affects people of all ages and is now one of the leading causes of death and disability.[25] In many countries with economies still in their formative years, such as China and India, diabetes has reached pandemic proportions. According to the World Health Organization (WHO), the rate of diabetes prevalence is increasing more quickly in low- and middle-income nations.[25]

Other risk factors that are associated with diabetes, such as population growth, unhealthy eating habits, and a sedentary lifestyle, also play an important role in the global increase in the diabetes epidemic. The rapid socioeconomic change, in conjunction with urbanization and industrialization, is the primary factor responsible for the global increase in the diabetes epidemic.[27,28]

Diabetes is a progressive illness that may lead to major problems. These complications are related with increasing expenditures for the diabetic patient's family, community, and the whole healthcare system. Uncontrolled diabetes puts patients at a higher risk of developing vascular disease, and the majority of the difficulties associated with type 2 diabetes are the result of complications related to the cardiovascular system (cardiovascular disease, cerebrovascular disease, and peripheral artery disease) and the microvascular system (diabetic retinopathy, nephropathy, and neuropathy).[27,28]

Pathogenesis

The onset of type 2 diabetes is brought on by a confluence of hereditary and environmental risk factors. These pathophysiological abnormalities are characterized by a malfunction of the beta cells, insulin resistance, and inflammatory processes, all of which eventually impair glucose homeostasis and lead to the development of microvascular and macrovascular disorders. In addition, these pathophysiological abnormalities are associated with an increased risk of developing type 2 diabetes. In patients with type 2 diabetes who also have insulin resistance, the

demand for insulin in tissues that are insulin targets is increased. In addition to being resistant to insulin, the pancreatic beta cells' activity was aberrant, which prevented them from meeting the increasing demand for insulin. This was a contributing factor in the development of diabetes.[29] On the other hand, the gradual loss of beta cells over time leads to a decrease in insulin production, which goes hand in hand with an increase in the need for insulin over time. It is possible that this will lead some people with type 2 diabetes who were previously insulin-independent to become insulin-dependent as a result of this. Because their insulin production continues and they very seldom become insulin deficient, the great majority of individuals who have type 2 diabetes do not need insulin treatment. This is because they very rarely run out of insulin.[30] When talking about hyperglycemia, the term "ominous octet" is often used to allude to the fact that there are at least eight different pathophysiological disorders that might contribute to impaired glucose homeostasis. This is because hyperglycemia can be caused by any one of these conditions.[31]

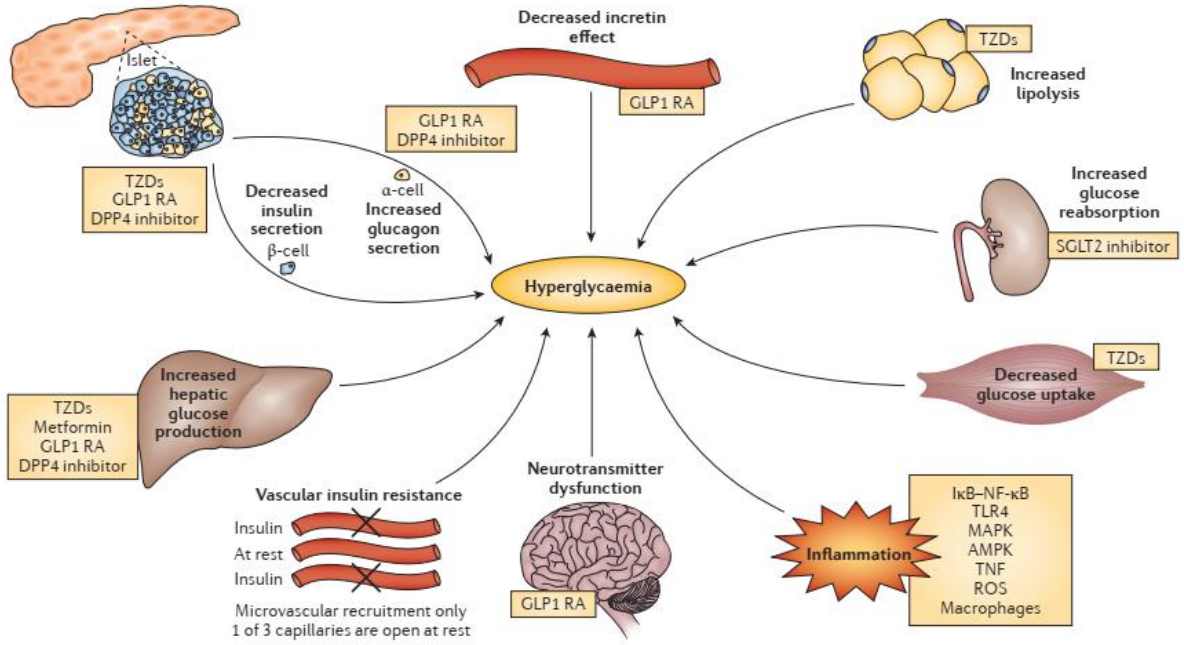


Figure 1: The ominous octet of hyperglycemia

Insulin resistance can show itself in a number of different ways in addition to diabetes. Some of these ways include obesity, nephropathy, essential hypertension, dyslipidemia (which is characterized by hypertriglyceridemia, low HDL, decreased LDL particle diameter, enhanced

postprandial lipemia, and remnant lipoprotein accumulation), ovarian hyperandrogenism and premature adrenarche, non-alcoholic fatty liver disease, and systemic inflammation. Insulin resistance can also be There is a correlation between insulin resistance and an increased likelihood of getting diabetes.[32]

Dyslipidemia in Diabetes

Diabetes almost often comes hand in hand with dyslipidemia. There is a correlation between the presence of atherosclerotic cardiovascular disease and elevated levels of blood cholesterol and triglycerides in individuals with type 1 and type 2 diabetes. Patients who have diabetes have a higher risk of coronary heart disease (CHD) for any given level of blood cholesterol, and the relationship between diabetes and hypertriglyceridemia is stronger than it is in the general population.[33] A decrease in circulating very-low-density lipoprotein (VLDL) and an increase in the catabolism of low-density lipoprotein (LDL) due to reduced glycation and upregulation of LDL receptors are the two mechanisms through which improved glycemic control produces beneficial effects on the levels of lipoproteins in diabetics. The reduction in cholesterol and triglyceride levels in diabetics is accompanied by this decrease.[34]

In patients with type 2 diabetes, dyslipidemia is characterized by elevated levels of triglycerides and lower levels of high-density lipoprotein (HDL) cholesterol. These changes are noticed several years prior to the start of clinically significant hyperglycemia. New data reveals that having a low HDL cholesterol level is not only a risk factor for cardiovascular disease but also for the development of diabetes in and of itself.[35] Even before diabetes is officially diagnosed, these alterations, together with the existence of tiny dense LDL particles, undoubtedly contribute to the acceleration of atherosclerosis.[36]

Pathophysiology

The underlying pathophysiology of diabetic dyslipidemia is complicated, and our understanding of it is not yet very advanced. Lipoprotein abnormalities in diabetes may be categorized into quantitative and qualitative. Quantitative alterations include a higher level of triglycerides and a lower level of HDL-C. Changes in the quality of the LDL include a rise in tiny dense LDL particles as well as a big very-LDL sub fraction (VLDL1), which is a factor that contributes to

the production of small dense LDL particles.[37] In insulin-resistant patients who are prediabetic and have normal glucose readings, hypertriglyceridemia, low HDL-cholesterol, and a preponderance of tiny dense LDL may be found years before the clinical diagnosis of type-2 diabetes. As a result, hyperglycemia cannot adequately explain the lipid alterations on its own. It is generally agreed that insulin resistance is the primary factor in the development of diabetic dyslipidemia.[38]

The presence of high levels of triglycerides in the blood is called hypertriglyceridemia. Triglyceride levels are important in identifying the typical lipid profile of diabetic dyslipidemia because of the crucial role hypertriglyceridemia plays in this process. Both fasting and non-fasting phases result in an increase in the creation of triglyceride-rich lipoproteins as well as a reduction in their clearance from the body. This leads to elevated triglyceride levels. One of the most noticeable characteristics of insulin resistance is an increase in the synthesis of very low-density lipoprotein, often known as VLDL. VLDL is the primary transporter of fasting triglycerides. Insulin has a role in every step of the generation and secretion of VLDL, according to Adiels.[39]

Insulin prevents lipolysis from occurring in adipose tissues by reducing the activity of hormone sensitive lipase. This enzyme is responsible for catalyzing the release of free fatty acids from triglycerides that have been accumulated. As a result, insulin is responsible for controlling the quantity of circulating free fatty acids, which serve both as substrates and regulatory factors throughout the process of VLDL assembly and secretion.[40] Insulin inhibits the transcription of microsomal triglyceride transfer protein in the liver. This protein is responsible for mediating the transfer of triglycerides to nascent apolipoprotein B (apoB), which is the primary surface protein of VLDL. Insulin is a hormone that regulates blood sugar levels. Because the pace at which apoB is produced is relatively stable, the quantity of apoB that is secreted is mostly governed by the rate at which it degrades, which in turn is determined by the degree to which lipidation occurs. Because of this, an increase in the availability of free fatty acids in the liver leads to a reduction in the breakdown of apoB, which in turn causes an overproduction of VLDL in conditions where insulin resistance is present.[41]

In addition to an increase in the synthesis of VLDL, hypertriglyceridemia is also caused by a reduction in the pace at which it is cleared from the body. A reduction in the clearance rate is related with diminished activity of lipoprotein lipase, a decrease in the hepatic absorption of VLDL, and an increase in postprandial triglyceride-rich chylomicrons. Lipoprotein lipase is an important enzyme in the delipidation cascade that occurs between VLDL and intermediate-density lipoprotein and LDL. Increased levels of free fatty acids may directly interfere with the action of lipoprotein lipase by inducing the enzyme to detach from the endothelium surface. This can have a negative impact on the lipoprotein lipase.[42]

Another symptom of diabetic dyslipidemia is postprandial hypertriglyceridemia, which is brought on by an increase in the synthesis of triglyceride-rich lipoproteins originating from the intestines as well as the liver. The synthesis rate of apoB-48 is sped up in patients with type 2 diabetes, and this acceleration corresponds with insulin levels.[43,44] The precise processes that are responsible for these modifications are not yet understood; nevertheless, it is possible that increased amounts of free fatty acids played a crucial impact. Intestinal lipoprotein synthesis may also be acutely boosted by monosaccharides, as this has recently been shown.[45] In addition, variations in the production and levels of incretins (glucagon-like peptide-1, glucagon-like peptide-2, and gastric inhibitory polypeptide) that are found in insulin resistance may potentially be responsible for inducing postprandial hypertriglyceridemia. Intestinal-derived chylomicrons and hepatic-derived VLDL compete for the same clearance route; as a result, increased levels of intestinal-derived chylomicrons may lead to a longer presence of hepatic-derived VLDL in plasma and vice versa. As a consequence of this, diabetes people have higher levels of triglycerides both when they are fasting and when they are not fasting.[39]

It is usual practice to connect an increase in triglyceride-rich lipoproteins with a decrease in levels of high-density lipoprotein (HDL) and an increase in levels of small dense LDL. The enzymatic activity of cholesteryl ester transfer protein (CETP) is stimulated when hypertriglyceridemia is present. CETP is the protein responsible for facilitating the transfer of triglycerides from triglyceride-rich lipoproteins to HDL and LDL in exchange for cholesteryl esters. This causes an increase in the amount of triglycerides that are included in both HDL and LDL. Because of this enhanced catabolism, triglyceride-rich HDL particles have a plasma half-life that is much shorter than other HDL particles. LDL particles that are rich in triglycerides are

subsequently hydrolyzed by lipoprotein lipase or hepatic lipase, which results in a decrease in the overall size of LDL particles.[46]

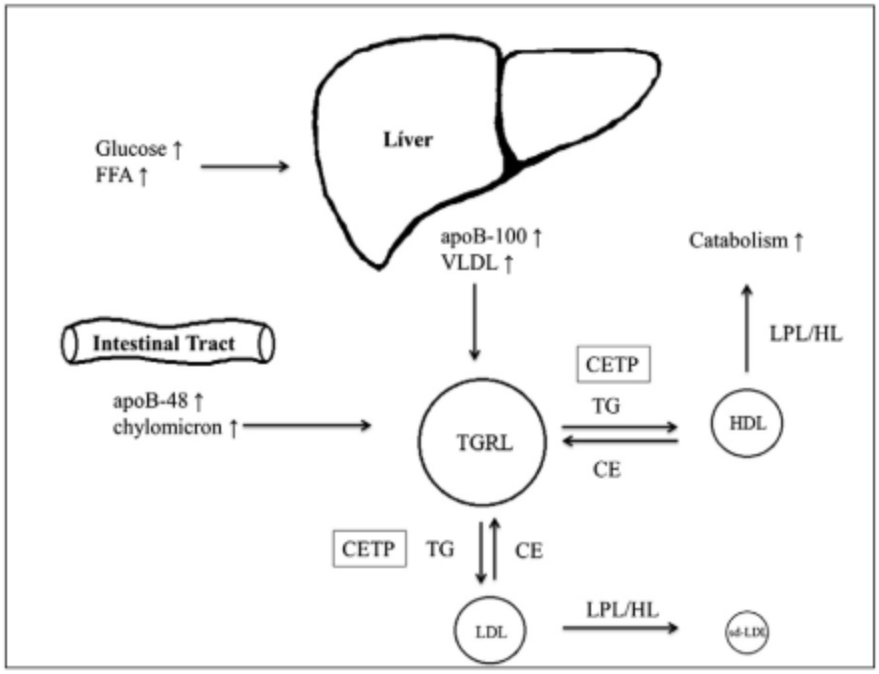


Figure 2: Triglyceride-rich lipoproteins, HDL, and LDL all change because of diabetes.

When glucose and free fatty acids are more available in the liver, apoB-100 is broken down less and VLDL is released more. When a person has just eaten, the intestinal tract makes more apoB-48 and chylomicrons. Triglyceride-rich lipoproteins in the blood are made up of VLDL from the liver and chylomicrons from the intestines. CETP makes it easier for LDL and HDL to trade triglycerides and cholesteryl esters with triglyceride-rich lipoproteins. Then, lipoprotein lipase or hepatic lipase breaks down the triglyceride-rich LDL and HDL.

Small dense LDL particles are more likely to undergo post-secretory changes such as glycation and oxidation. These particles also infiltrate the intima more readily, where proteoglycans bind them and prevent them from circulating. Consequently, while there is not a significant increase in LDL cholesterol, there is a rise in the number of tiny dense LDL particles, which are more atherogenic. In addition, people with type 2 diabetes have an increase in the amount of atherogenic particles called chylomicrons and VLDL remnants.[37,47]

In conclusion, diabetics have post prandial lipemia, elevated levels of VLDL and small dense LDL, and decreased levels of HDL. All of these elements play a part in the link between diabetic dyslipidemia and atherosclerosis, and it's a role that's extremely significant.

Diabetic Dyslipidemia and Atherosclerotic Cardiovascular Disease

It is widely recognized that a causal association exists between LDL and atherosclerosis, however this does not hold true for HDL.[48] Triglyceride-rich lipoproteins seem to have a causative role in cardiovascular events, according to more recent research.[49] Patients diagnosed with type 2 diabetes may not always have a greater LDL concentration compared to persons who do not have diabetes. On the other hand, there is a discernible rise in the number of tiny dense LDL particles, which indicates that diabetes individuals have a bigger number of LDL particles while having a same LDL cholesterol content. When compared to big buoyant LDL particles, small dense LDL particles are more atherogenic because they are more likely to be eliminated by scavenger receptors, which is an early crucial event in the development of atherosclerosis.[50] Even among diabetic people whose LDL levels are lower than the threshold set by the National Cholesterol Education Program of 130 mg/dl, the amount of LDL cholesterol continues to be a significant independent predictor of cardiovascular disease (CVD). According to the findings of the Strong Heart Study, the risk of cardiovascular disease rose by 12% in diabetes participants for every 10 mg/dl rise in LDL cholesterol.[51]

The fact that high triglyceride levels are often linked with simultaneous alterations in HDL and LDL levels is the primary factor that contributes to the complexity of isolating the influence of hypertriglyceridemia on cardiovascular disease (CVD). Triglycerides do not have a direct role in the progression of atherosclerotic lesions. This is due to the fact that the free fatty acids that are produced from triglycerides serve either as an active energy source or a stored energy reserve. Triglycerides, on the other hand, are carried in lipoproteins that contain apoB, and these lipoproteins have the potential to promote atherosclerosis in a manner similar to that of LDL. In addition, non-fasting triglyceride levels may be used as a surrogate marker for non-fasting residual cholesterol levels.[52] Varbo et al. studied the causal association between increased residual cholesterol levels and risk for coronary heart disease by using data from three major studies that were conducted in Denmark and had a combined total of 73,513 participants.

According to the findings of the research, an increase of 1 mmol/l in non-fasting residual cholesterol was related with a 2.8-fold increase in the causative risk for ischemic heart disease. This was true despite the fact that HDL levels were reduced.[53]

The precise function that HDL plays in cardiovascular disease has been a mystery for a very long time. HDL has been considered the "good" lipoprotein for a long time since epidemiological and clinical investigations have shown an inverse correlation between HDL concentration and the risk of cardiovascular disease (CVD).[54] The most significant role of HDL in preventing atherosclerosis is the transfer of cholesterol in the opposite direction.[55] HDL also demonstrates additional possible cardioprotective properties, including anti-oxidative, anti-inflammatory, and endothelium-dependent vasodilatory actions. These functions are all carried out by HDL. It has not been possible to obtain a consistent and meaningful decrease in cardiovascular events with the use of pharmaceutical methods to increase HDL.[48,56] The seeming contradiction shows that HDL concentrations on their own are not sufficient to completely explain the cardioprotective benefits that epidemiological studies have shown to exist. It has been hypothesized that HDL concentration could be nothing more than a surrogate indication of HDL functioning and/or the concentration of triglyceride-rich apoB-containing lipoproteins. Because HDL cholesterol is a sensitive marker for an elevated concentration of atherogenic triglyceride-rich lipoproteins, a decrease in HDL cholesterol concentration is associated with type 2 diabetes. This reduction in HDL cholesterol concentration is indicative of an increased risk of cardiovascular disease (CVD). In addition to this, HDL function is significantly compromised, which further raises the risk of cardiovascular disease.[3,57]

Glycosylated Haemoglobin (HbA1c)

Glucose and the amino-terminal valine of one or both beta chains of HbA interact in a multistep condensation reaction with an amine moiety of hemoglobin to form the specific glycated hemoglobin (GHB) known as HbA1c. This reaction takes place after glucose has condensed with an amine moiety of hemoglobin.[58] Boronate affinity chromatography was not able to assess these additional glycated components, in addition to HbA1c, until the early 1980s. Prior to this time, HbA1c was the only glycated component that could be detected.[59] GHBs, including HbA1c, are continually produced from HbA at a rate that is proportional to the concentration of glucose in

the surrounding environment. Therefore, the quantity of GHB is determined by the lifespan of the erythrocyte as well as the quantities of glucose that the erythrocytes have been exposed to over their lifetime. Since HbA1c is a "weighted" average of blood glucose levels over the previous 120 days, it follows that glucose levels within the previous 30 days contribute a great deal more to the overall level of HbA1c than glucose levels over the previous 90–120 days.

The significance of HbA1c as an indication of both mean glycemia and the associated outcome risks was not conclusively determined until the findings of the Diabetes Control and Complications Trial (DCCT) were made public in 1993. Prior to this time, the value of HbA1c as an indicator remained unclear.[60,61] The Diabetes Control and Complications Trial (DCCT) was a large prospective long-term randomized trial that included 1441 patients who were followed for a mean of 6.5 years. This trial offered conclusive proof that intensive glycemic control significantly reduces the risk of long-term diabetes complications and permitted the establishment of specific HbA1c treatment goals. However, practical implementation of suggested HbA1c goals that were based on these research was problematic since there was a lack of comparability of HbA1c values across test techniques and labs. These investigations were conducted in the United States.[62] Therefore, in 1996, the National Glycohemoglobin Standardization Program (NGSP) was developed with the purpose of standardizing HbA1c findings to resemble the results of the DCCT/UKPDS. This was done in an effort to diagnose the disease early.[62,63]

Random blood sugar (RBG), fasting blood sugar (FBG), and glycosylated hemoglobin are the three components that are used in the process of diagnosing hyperglycemia (HbA1c). Out of them, the HbA1c level is the one that is generally accepted as the gold standard for regular indicators of glycemic management. Insulin resistance, hyperglycemia, dyslipidemia, hypercoagulability, and inflammation are some of the most important pathogenic processes that are associated with increased HbA1c values. The degree to which haemoglobin is glycosylated is directly proportional to the amount of glucose present in the blood. A high quantity of glycohaemoglobin suggests that the cell has been exposed to a significant amount of glucose during its lifetime. Therefore, the concentration of HbA1C is connected to the levels of glucose in the blood over a period of time, and this gives an assessment of the patients' long-term management and degree of control. Individuals who have poor glycemic control may be

identified with a single HbA1c test. Once these patients have been identified, lifestyle changes and pharmaceutical interventions can be implemented to enhance the control. After then, more testing will be necessary until the desired HbA1c level is reached.[64,65]

Limitations of HbA1c Testing

HbA1c is a great indicator of the degree to which one has their diabetes under control for the overwhelming majority of diabetic people. In a similar vein, the HbA1c test can be used to diagnose diabetes in the vast majority of patients. However, the HbA1c test may not be trustworthy in some circumstances. Such situations will impact HbA1c findings regardless of the assay methods used. These conditions include severe iron deficiency anemia and recent blood transfusions. Renal anemia with erythropoietin usage and hemolytic anemia are two examples of such disorders. In addition, variations in hemoglobin or adducts attached to hemoglobin might result in method-specific interferences.[66] Researchers have also discovered that there is some degree of HbA1c variability across individuals, and that this variation may be attributed to a variety of different possible variables such as age or ethnicity. In situations of renal failure, HbA1c test results may be 1–2% lower than they would be in individuals whose kidney function is normal. This is one of the most prevalent disorders that may impact the interpretation of HbA1c test results.[67] In light of the fact that it is essential to have sufficient glycemic control in these patients, an incorrect interpretation of the data might result in a larger risk of complications than was originally anticipated. Iron deficiency anemia is another issue that is rather common in poor nations and seems to contribute to artificially high HbA1c findings. Iron replacement treatment brings HbA1c levels back down to the levels that are considered normal for a person with diabetes.[66,68]

Association of HbA1c with Lipid profile

Hyperglycemia, dyslipidemia and coronary artery disease relate well with each other in type 2 diabetes and it has been proposed that higher prevalence of cardiovascular disease in type 2 diabetes is due to chronic uncontrolled hyperglycemia. It has been observed that the risk of cardiovascular disease increases by 18% for every 1% increase in value of HbA1c in diabetics. Studies have suggested that HbA1c is an autonomous risk factor for cardiovascular disease and stroke.[65] It should be understood that HbA1c cannot replace the utility of lipid profile, but if

presence of certain correlation is discovered between the two, HbA1c could be considered for early determination of dyslipidemia and hence could help in assessing CVS risk. However, there are several reports too indicating that there is a discrepancy regarding the relationship between HbA1c and the lipid profile.[69]

The levels of glycated haemoglobin (HbA1c), fasting blood glucose (FBG), total cholesterol (TC), low density lipoprotein (LDL), high density lipoprotein (HDL), and triglyceride (TG) were measured in 132 patients by Ozder et al.[70] They discovered that serum levels of triglycerides, cholesterol, low-density lipoprotein, and hepatosteatosis were significantly correlated with HbA1c (p 0.05).

In individuals who were suffering from diabetes mellitus, Sati et al. investigated whether or not there was an association between dyslipidemia and glycemic indicators such as fasting blood glucose, glycated hemoglobin, and fasting insulin levels. Patients suffering from Type II diabetes made up the total number of participants in the research. The levels of glycated haemoglobin (HbA1c) were utilised as the criteria for categorising the participants into the three different groups. Group A patients exhibited HbA1c values that were less than or equal to 7%, Group B patients had values that ranged from 7-9%, and Group C patients had HbA1c values that were greater than or equal to 9%. The patient's Body Mass Index (BMI) ranged from 26 to 33, placing them in the category of individuals who are both overweight and obese. The progression of dyslipidemia from Group A to Group C was accompanied by an increase in the levels of fasting blood sugar, HbA1c, and HOMA-IR, as determined by multivariate analysis (Homeostatic Model Assessment of Insulin Resistance). A bivariate correlation analysis was performed on the data from all of the patients, and it was discovered that there is a direct positive correlation between the values of High-density lipoprotein-C and Fasting blood sugar, Glycated haemoglobin, duration of disease, and HOMA-IR values. This correlation was found by looking at the data from all of the patients. The significance level for this correlation was set at less than 0.05. There was a substantial correlation between bad lipid profiles and glycated hemoglobin, fasting blood sugar, and insulin resistance.[71]

Panjeta et al. investigate the lipid profile in the blood of diabetic individuals with type 2 diabetes, as well as the correlation between the lipid profile and HbA1c levels. Participants in the

observational cross-sectional research ranged in age from 32 to 94 years old and had diabetes. There were 30 male and 30 female diabetic patients in the study. Patients were separated into two groups based on their HbA1c levels: Group 1 had a HbA1c level of 7%, which is regarded to have good glycemic control, and Group 2 had a HbA1c level that was greater than 7%. (poor regulation of glycemic levels). We determined the glucose concentration, the HbA1c level, and the lipid profile, which was broken down into total cholesterol levels, triglyceride levels, low-density lipoprotein levels, and high-density lipoprotein levels (HDL). In the group of patients who had excellent glycemic control, significantly lower values of glucose concentration, total cholesterol, and the ratio of total cholesterol to HDL-C were found. ($p < 0.0005$). According to the findings of our research, there is a substantial positive link between HbA1c and triglyceride level ($r = 0.375$; $p = 0.003$) as well as HbA1c and the ratio of triglyceride to HDL-C ($r = 0.335$; $p = 0.009$). According to the findings of the research, the HbA1c test might be utilized not only as a glycemic control metric but also as a predictor of dyslipidemia in type 2 diabetes patients.[72]

Mohammed and colleagues (2020) investigated whether or whether patients with type 2 diabetes mellitus had an association between their lipid profiles and their levels of glycated hemoglobin (HbA1c). 31% of participants had a body mass index (BMI) that was higher than 30 kg/m2, 65% of participants had a waist-to-hip ratio that was larger than 0.95, and 76% of participants had a waist-to-height ratio that was greater than 0.55. The average level of glycation in the hemoglobin was 8.76 percent. Patients whose HbA1c was more than 7% had significantly higher BMIs, higher rates of high-speed serum triglycerides and high-density LDL particles, as well as higher rates of high-speed plasma glucose, in comparison to patients whose HbA1c was less than 7%. Glycated Hb was shown to have a correlation with total cholesterol and triglyceride levels, but age, BMI, total cholesterol, and HDL-C levels were not found to have significant relationships.[69]

In individuals with type-2 diabetes mellitus, Ahmad et al. investigated whether or not there was an association between glycated haemoglobin and dyslipidemias (DM). There were a total of 60 patients diagnosed with type-2 diabetes and 40 healthy controls who were matched for age and gender. After an overnight fast, a glycated haemoglobin (HbA1c) test, a fasting blood glucose (FBG) test, and a lipid profile were carried out. The group serving as a control was referred to as Group-A, while the group of patients was referred to as Group-B. There was a positive correlation between HbA1c and all of the values of the lipid profile, with the exception of HDL-

C, which had a negative correlation. The researchers came to the conclusion that there was a link between the degree of glycemic control (measured by HbA1c) and the severity of dyslipidemia in type 2 diabetes patients.[73]

Dash investigated the extent to which individuals with type 2 diabetes had an association between their HbA1C levels and their lipid profiles. The FBS, PPBS, HbA1c, TG, Chol, HDL, LDL, and VLDL values were analyzed and compared between the case group and the control group. The case group had substantially elevated levels of FBS, PPBS, and HbA1C. When compared to the control group, the case's levels of LDL, CHOL, and TG were considerably higher than those of the control group, while HDL levels were significantly lower. There was a strong correlation found between HbA1c and lipid profiles. According to the findings of the research, people with type 2 diabetes were at a greater risk of having dyslipidemia. There was a strong correlation found between HbA1c and lipid profiles. Therefore, in addition to its function as a biomarker for glycemic management, HbA1c may also serve as an indirect predictor of dyslipidemia in patients with type 2 diabetes.[74]

Purohit et al. identified lipid abnormalities linked to type 2 diabetes mellitus and found a relationship between elevated levels of glycated hemoglobin (HbA1c). In the cross-sectional research, a random sample of 140 people who were already diagnosed with Type-2 Diabetes Mellitus was taken. Their HbA1c and lipid profiles were analyzed as part of the investigation. HbA1c correlated strongly with total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), triglycerides (TG), the Tc/HDL ratio, and non HDL-cholesterol. In a similar manner, researchers discovered that HbA1C and HDL cholesterol have a highly significant inverse association with one another. They reasoned that HbA1c may be utilised as an indicator of dyslipidemia, and that early detection of dyslipidemia might be employed as a prophylactic intervention for the improvement of CVD in patients with type 2 diabetes, in addition to providing reliable glycemic control.[75]

Patients with type 2 diabetes mellitus were studied by Alzahrani et al. (2019), who looked at the relationship between glycated hemoglobin (HbA1c) and the lipid profile (T2DM). 141 females and 65 males were chosen to participate in the study of type 2 diabetes patients. The participants in the research were divided into two categories according to their HbA1c readings: those with a

good glycemic index of less than 7% and those with a bad glycemic index of more than 7%. When HbA1c and the other parameters were analyzed for their association, the results showed that there was a significant link between HbA1c and TG (r=0.16, p=0.020), but there was no significant relationship with the other variables. Linear regression study revealed a correlation between HbA1c and TG (p=0.020), and this relationship held true after controlling for other variables.[76]

MATERIALS AND METHODS

Type of Study: A hospital based cross-sectional observational study.

Study Setting: The study was conducted in Department of Medicine, Santosh Medical College and hospital, Ghaziabad on patients who will be attending the Medicine OPD of Santosh Hospital.

Study Period: The study period is conducted for a period of one year after getting approval of the Institutional Human Ethics Committee.

Study population: The study population includes patients presented to Department of Medicine in the hospital; fulfilling the below mentioned inclusion and exclusion criteria.

Inclusion criteria:

1. Age group ≥ 18 years of either sex.
2. Patient with type II DM (as per American Diabetes Association).
3. Patients of T2DM and suffering with the disease for a minimum of 1 year.
4. Patient with written informed consent by patient/guardian.

Exclusion criteria:

1. Any patient suffering from any other metabolic disease or disorder other than Type-2 Diabetes Mellitus.
2. Patients suffering from CRF (chronic renal failure) or liver function impairment or any other disease.
3. Those receiving lipid altering drugs

METHOD OF SAMPLE SIZE CALCULATION:

The required sample size for the current study was calculated with reference to an article published by Aishwarya Purohit et al., in 2017 in Scholars Journal of Applied Medical Sciences. The Sample size was calculated on the basis of following formula:

$SS = 0.5\ Z_n\ (1+r/1-r)$

- SS = Sample Size
- Z-score = critical value for standard normal distribution

  $Z_n = [\{Z_\alpha + Z_\beta\} + c]^2 + 3$

  $Z_\alpha = 1.96$ at $\alpha = 5\%$

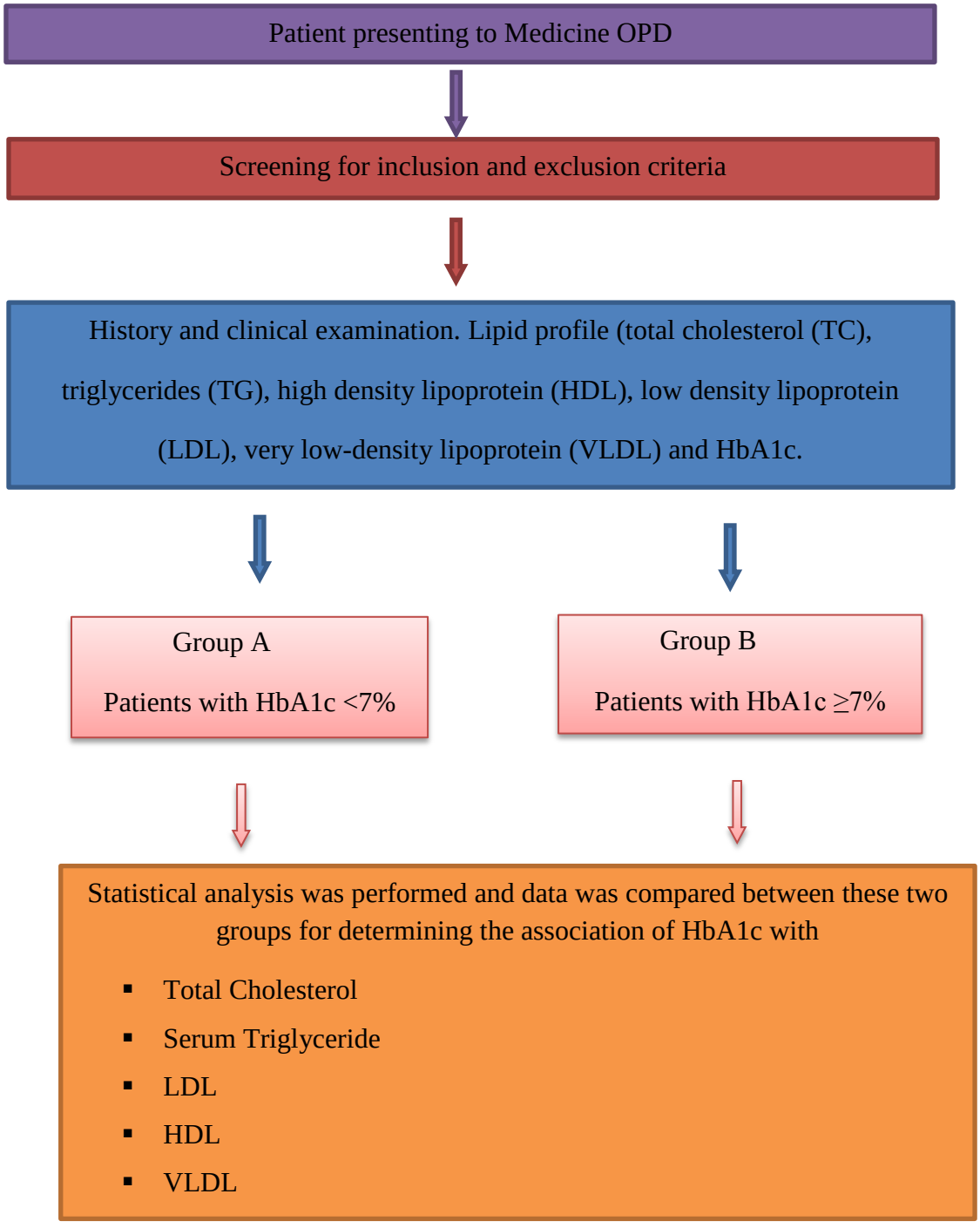
  $Z_\beta = 0.84$ at 80% [Power of test]
- r = Correlation coefficient (0.16)

Sample Size = 290 consisting of Group A= 145 and Group B= 145.

Method of Study:

A predesigned, self-administered proforma is designed keeping the objectives of the study at the center point. The purpose of the study was explained to the patient and informed consent were obtained. Patients were selected for study which satisfied all criteria. In the construction of the proforma, utmost care was taken to make it broad based, so that all the aspects desired to be studied could be incorporated in its body. Demographic information of the participants including age, sex, smoking status and personal medical history was recorded. The health examination in order to collect the anthropometric measurements, including weight and height were used to calculate the body mass index (BMI). Weight was measured using an electronic digital scale, and height was measured using a wall-mounted stadiometer. BMI was calculated as weight (Kg) per height squared (m^2). Patients with an established diagnosis of T2DM were selected according to the American Diabetes Association criteria established in 2007. These criteria set the following as values that are indicative of T2DM: HbA1C ≥6.5%, FPG ≥126 mg/dl (7.0 mmol/l), 2-h

plasma glucose ≥200 mg/dl (11.1 mmol/l) during an oral glucose tolerance test (OGTT), or random plasma glucose ≥200 mg/dl (11.1 mmol/l).


Patient presenting to Medicine OPD
Screening for inclusion and exclusion criteria
History and clinical examination. Lipid profile (total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL), low density lipoprotein (LDL), very low-density lipoprotein (VLDL) and HbA1c.
Group A
Patients with HbA1c <7%
Group B
Patients with HbA1c ≥7%
Statistical analysis was performed and data was compared between these two groups for determining the association of HbA1c with
Total Cholesterol
Serum Triglyceride
LDL
HDL
VLDL

Subject Confidentiality & Consent:

Data collection was on paper. The patients participating in the study were contacted only by the persons involved in the study. All the patient specific data was kept in strict confidence. Patient identifiable data (name, contact, address, etc.) has not been presented in journal or any public forum. Informed consent process was initiated prior to the individual agreeing to participate in the study and continuing throughout the individual's study participation. The form has provide all the information required for the patient to understand their role in the study. It had contain a description of the study procedures. It was in a language understandable by each member of the study population. It was submitted for the ethical committee clearance.

Financial Inputs and Funding:

All the study related procedures and investigations were according to the institutional protocol for the management of desired surgeries. All these expenses were borne by the patient himself/herself. Also this study was not being sponsored by any pharmaceutical company.

Ethical Considerations:

The protocol of the present study has been submitted to the Ethics Committee of Santosh Medical College & Hospital for review. After getting their approval the study was initiated in the institution. Also prior to enrolling any patient in the study, a voluntary written informed consent was obtained for participation. This consent was in addition to the other consents which were obtained as per the institutional protocol.

STATISTICAL ANALYSIS

The data was collected using a predesigned template which was filled by the concerned physician involved in the immediate treatment of the patient. The collected data variables obtained were compiled by using an excel spreadsheet. Continuous variables like age, lipid function test, HbA1c, and duration of diabetes were described as mean ± SD unless otherwise stated, while categorical variables like gender, risk factor history like smoking, comorbidities like peripheral arterial disease and hypertension and lifestyle were presented in frequencies and percentages. After appropriate data filtration, the data sheet was transferred and analysed using Graphpad Prism. Pearson correlation test (r) was used to evaluate the relationship between HbA1c and triglycerides. Chi-square test was used for association between HbA1c with various cut-off values and high triglyceride levels.

Significant figures:

** Strongly significant (p value: $p \leq 0.01$)

* Moderately significant (p value: $0.01 < p \leq 0.05$)

+ Suggestive significance (p value: $0.05 < p < 0.10$)

NS- Non-significant (p value: $p > 0.1$)

OBSERVATIONS AND RESULTS

We evaluated 290 patients for our study. The patients were divided into two groups, control (HbA1c<7%) and cases (HbA1c>7%). The study had quite similar distribution amongst the age groups with maximum patients between 41 to 50 years of age. The mean age group of the control group was 53.61 ± 10.86 while that of the cases was 51.06 ± 10.32. The mean age of the entire study population was 52.33. The details are depicted in the table and Figure below

Table-1: Distribution of patients according to age in cases and control group

		GROUP						Chi-Square	p-value
		Control		Cases		Total			
AGE	<=40	20	13.8%	25	17.2%	45	15.5%	3.507	.320
	41-50	43	29.7%	53	36.6%	96	33.1%		
	51-60	41	28.3%	37	25.5%	78	26.9%		
	>60	41	28.3%	30	20.7%	71	24.5%		
	Total	145	100.0%	145	100.0%	290	100.0%		
	Mean	53.61		51.06		52.33			
	SD	10.86		10.32		10.65			

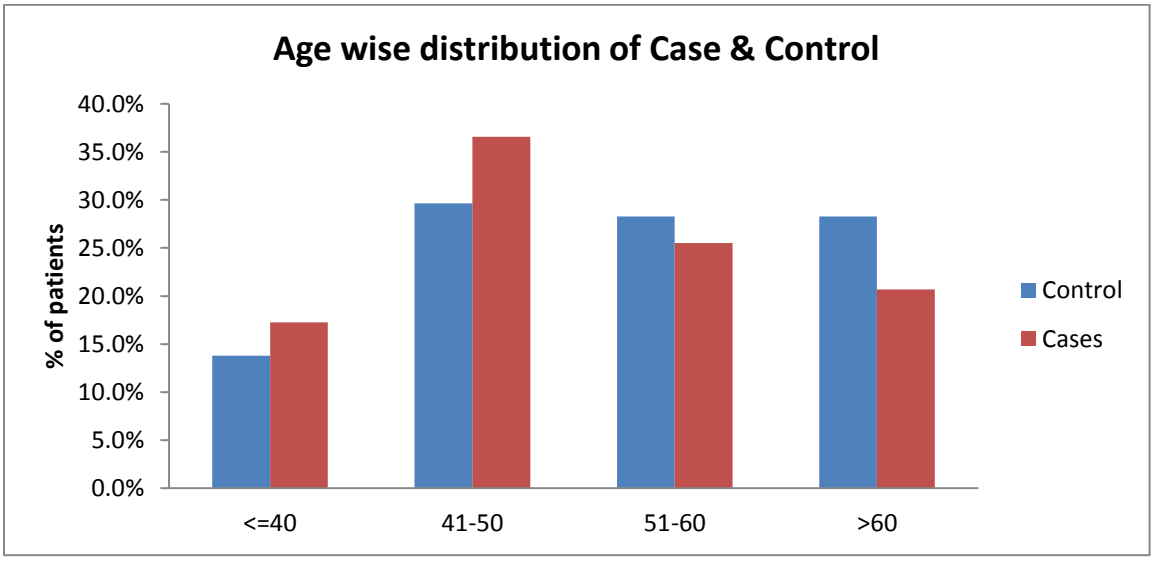


Fig. 1: Distribution of patients according to age in cases and control group

The gender distribution had a female predominance in our study with 54.8% females. The distribution amongst the groups was also similar with 66 males in control group and 65 in the cases group. The details are depicted in the table and Figure below.

Table-2: Distribution of patients according to gender in cases and control group.

		GROUP						Chi-Square	p-value
		Control		Cases		Total			
SEX	Male	66	45.5%	65	44.8%	131	45.2%	.014	.906
	Female	79	54.5%	80	55.2%	159	54.8%		
	Total	145	100.0%	145	100.0%	290	100.0%		

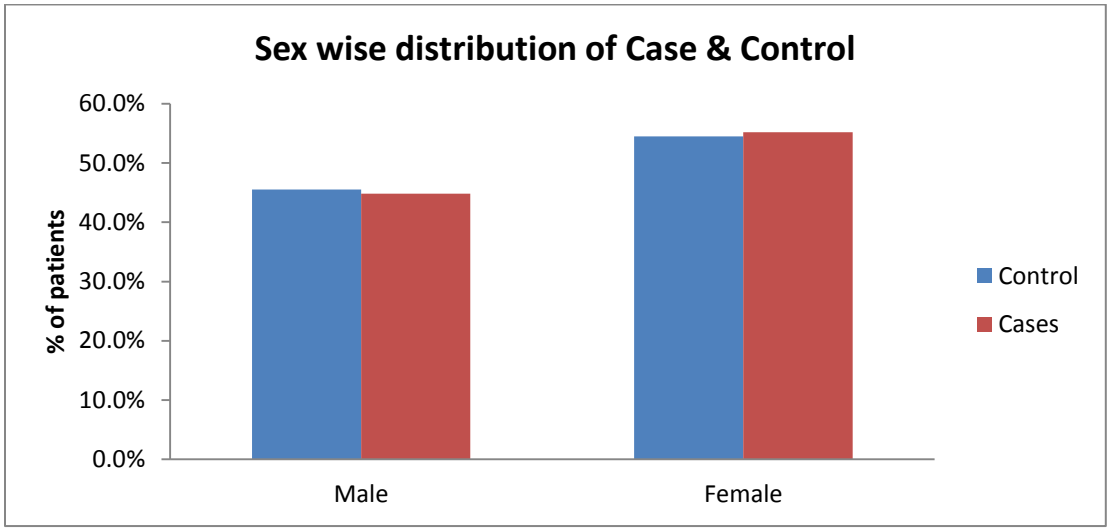


Fig. 2: Distribution of patients according to gender in cases and control group.

The mean HbA1c of the control group was 6.21 ± 0.34 while that of the cases was 9.47 ± 1.47. The difference was statistically significant with a p-value of 0.0001. The difference is expected since the cases had HbA1c more than 7 and controls had HbA1c less than 7. The details are depicted in the table and Figure below

Table-3: Mean value of HbA1c among cases and control group.

GROUP		N	Mean	Std. Deviation	t-value	p-value
HBA1C	Control	145	6.21	0.34	26.008	.0001**
	Cases	145	9.47	1.47		

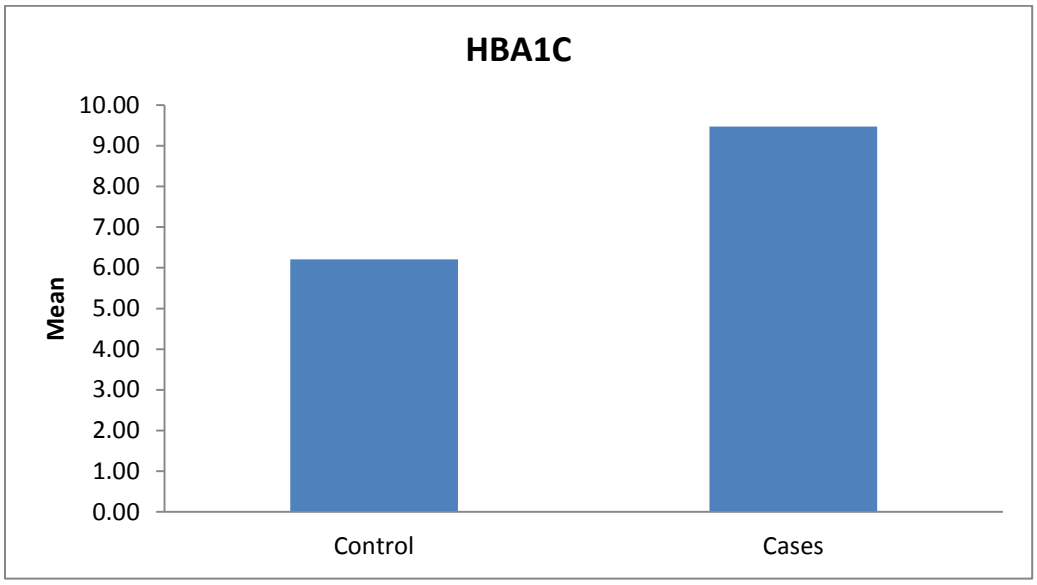


Fig. 3: Mean value of HbA1c among cases and control group.

We also observed a similar difference between the mean FBS and mean PPBS values between the two groups. The mean FBS was significantly lower in controls than cases and the same can be said about the mean PPBS. The following table and figure depicts the story. The details are depicted in the table and Figure below

Table-4: Mean value of FBS and PPBS among cases and control group.

GROUP		N	Mean	Std. Deviation	t-value	p-value
FBS	Control	145	112.82	36.97	10.903	.0001**
	Cases	145	176.66	60.04		
PPBS	Control	145	158.67	62.16	13.712	.0001**
	Cases	145	264.11	68.63		

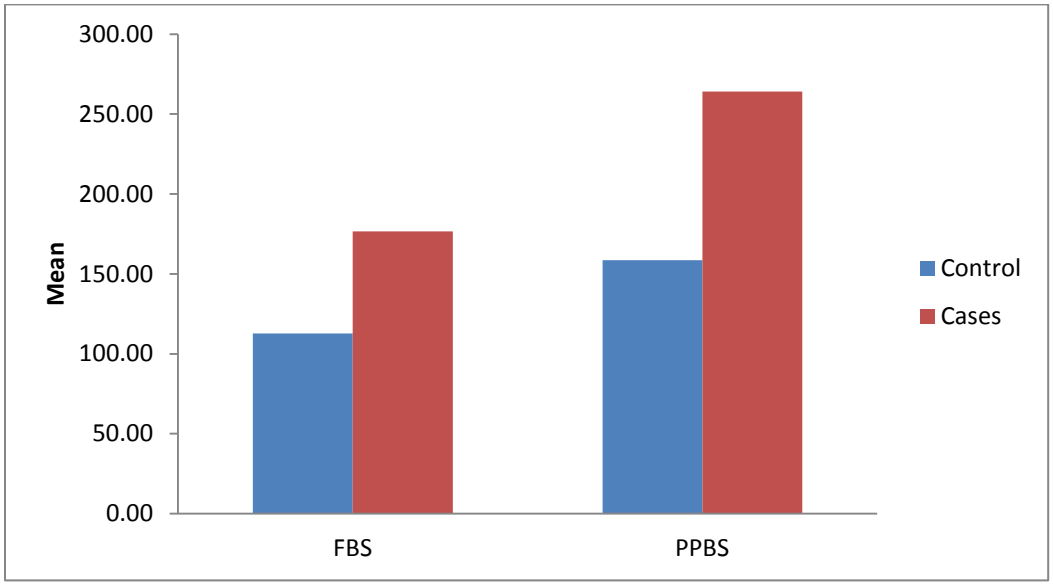


Fig. 4: Mean value of FBS and PPBS among cases and control group.

The lipid profile followed a similar trend as of the blood sugar values. The total cholesterol, low density lipoprotein, the very low density lipoprotein and the triglycerides were significantly lower in the control group. The p value was 0.0001 for these four parameters and the difference was statistically significant. The good cholesterol or high density lipoprotein was higher in controls and the difference was again statistically significant. The respective values are mentioned in the table depicted below. The details are depicted in the table and Figure below

Table-5: Mean values of lipid profile among cases and control group.

GROUP		N	Mean	Std. Deviation	t-value	p-value
TC	Control	145	161.76	30.40	9.510	.0001**
	Cases	145	216.24	61.92		
HDL	Control	145	50.52	13.00	3.950	.0001**
	Cases	145	44.44	13.20		
LDL	Control	145	103.84	20.37	5.779	.0001**
	Cases	145	127.54	44.97		
VLDL	Control	145	35.16	13.60	7.217	.0001**
	Cases	145	47.86	16.26		
TRIGLY	Control	145	138.76	36.47	4.677	.0001**
	Cases	145	167.94	65.69		

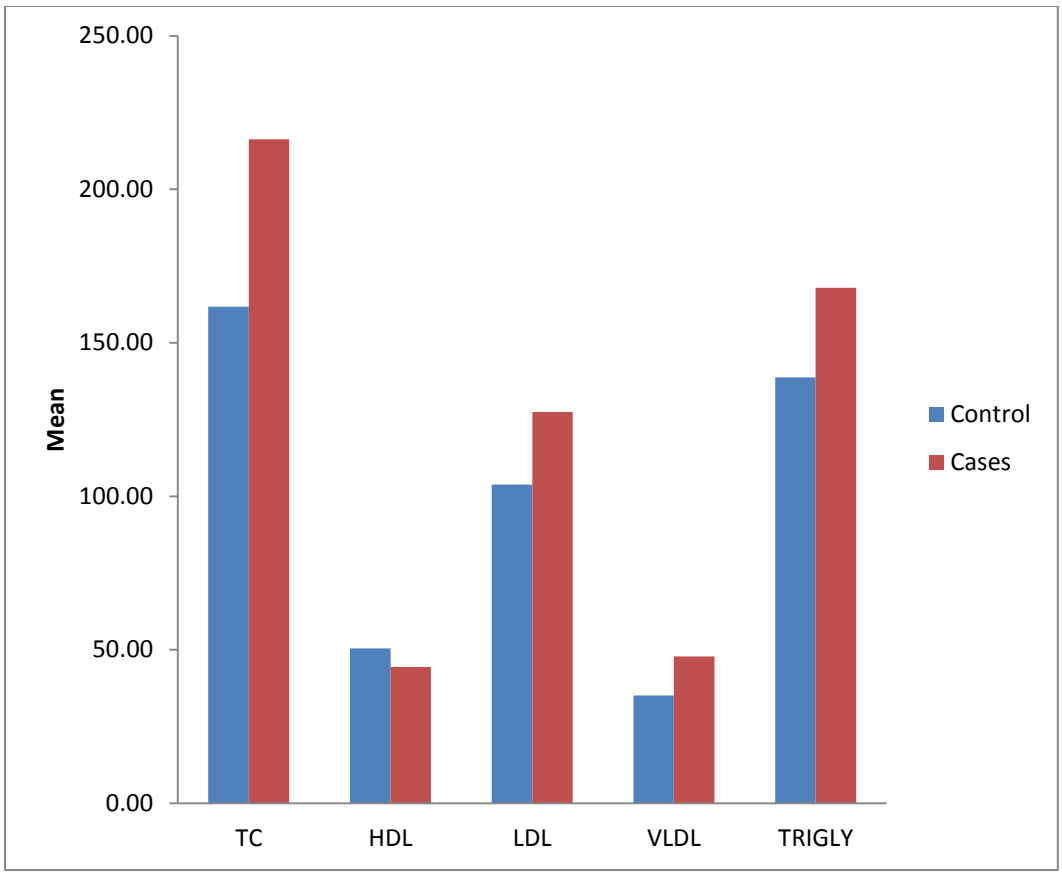


Fig. 5: Mean values of lipid profile among cases and control group.

The mean BMI in the control group was 23.57 ± 2.54, which increased to 26.86 ± 3.66. The difference here was again statistically significant. The details are depicted in the table and Figure below

Table-6: Mean value of BMI among cases and control group.

GROUP		N	Mean	Std. Deviation	t-value	p-value
BMI	Control	145	23.57	2.54	8.907	.0001**
	Cases	145	26.86	3.66		

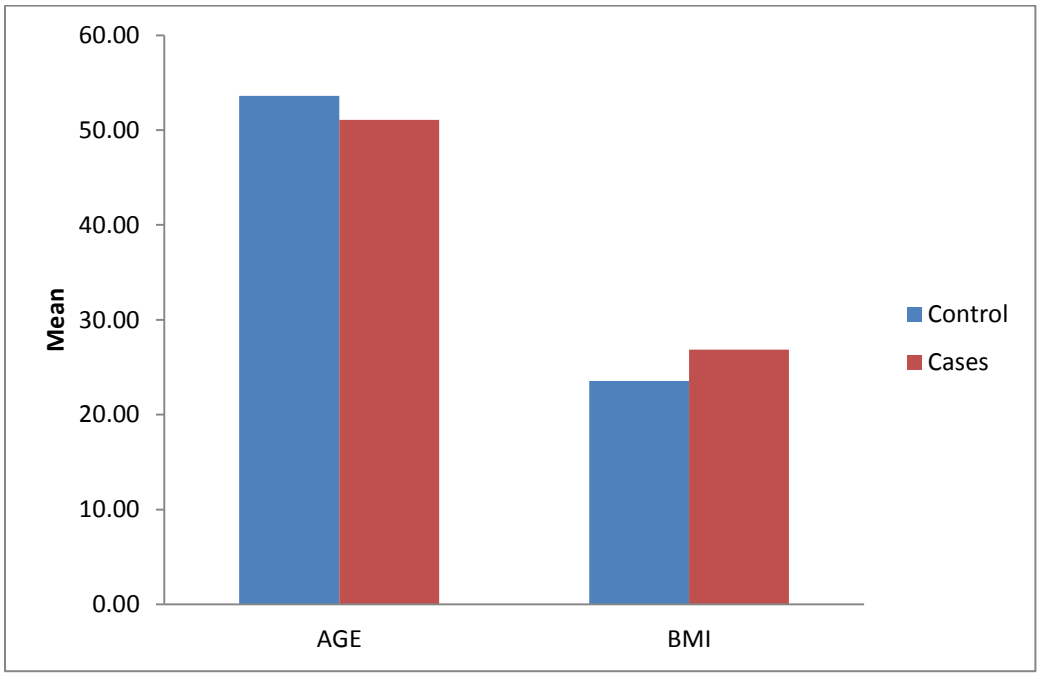


Fig. 6: Mean value of AGE and BMI among cases and control group.

When we checked the correlations of HbA1c with our various parameters, we observed that it had a positive correlation with every parameter except for HDL. All correlations were statistically significant with a p-value of 0.0001 or less. The table below highlights the correlations with highest Pearson's correlation co-efficient of 0.704 with PPBS and the lowest Pearson's correlation co-efficient of 0.309 with triglycerides. The details are depicted in the table and Figure below

Table-7: Correlations of HbA1c with various parameters.

Correlations			
	HBA1C		
	Pearson Correlation	p-value	N
FBS	.645**	.0001**	290
PPBS	.704**	.0001**	290
TC	.542**	.0001**	290
HDL	-.223**	.0001**	290
LDL	.423**	.0001**	290
VLDL	.391**	.0001**	290
TRIGLY	.309**	.0001**	290
BMI	.474**	.0001**	290
**. Correlation is significant at the 0.01 level (2-tailed).			
*. Correlation is significant at the 0.05 level (2-tailed).			

The FBS had a Pearson's correlation co-efficient of 0.645 while that of PPBS was 0.704.

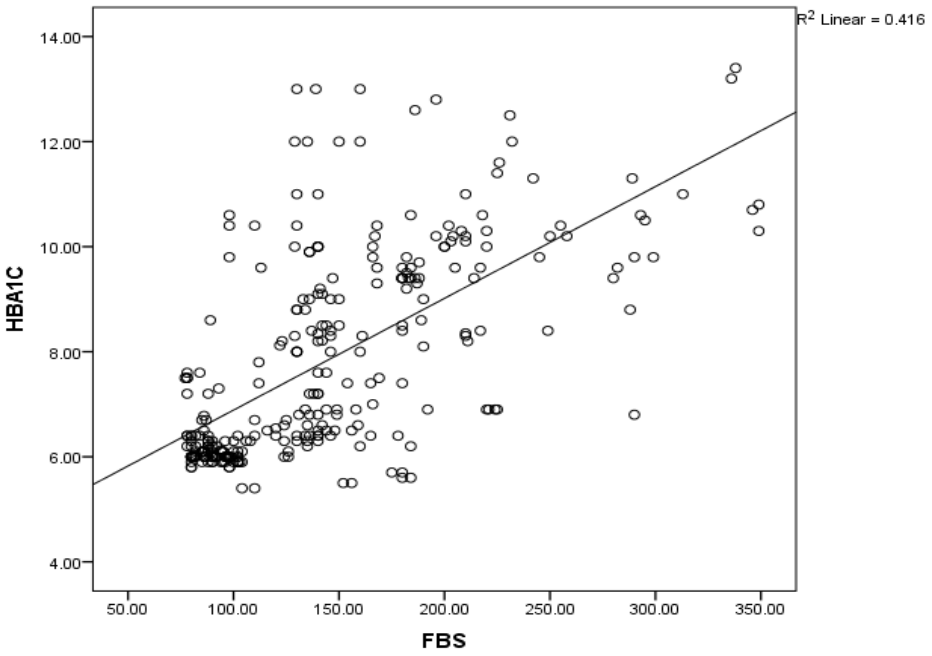


Fig. 6: Correlations of HbA1c with FBS.

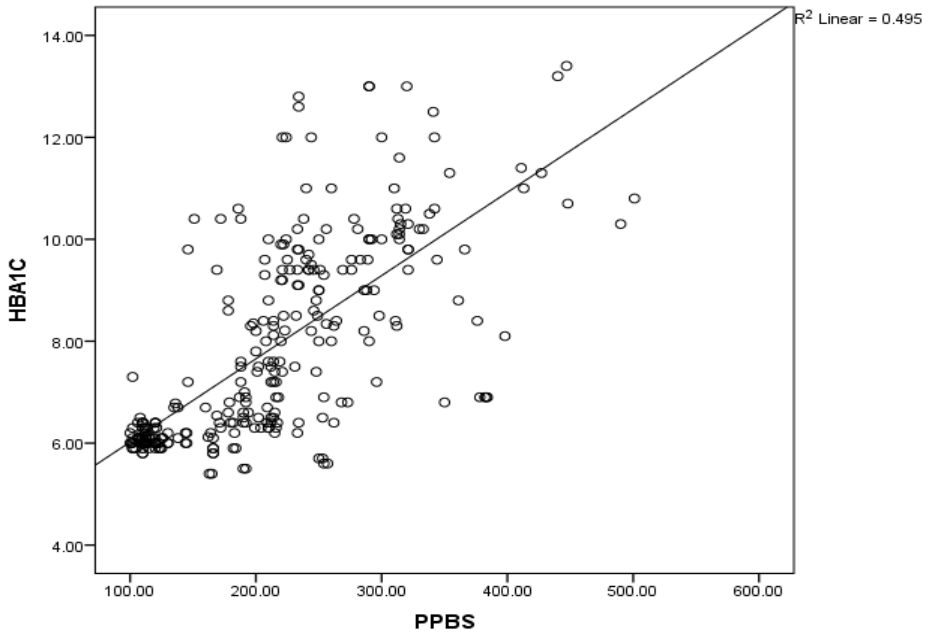


Fig. 7: Correlations of HbA1c with PPBS.

The total cholesterol, LDL, VLDL and triglycerides had a positive correlation with 0.542, 0.423, 0.391 and 0.309 respectively while HDL had a negative correlation with Pearson's correlation co-efficient value of 0.223

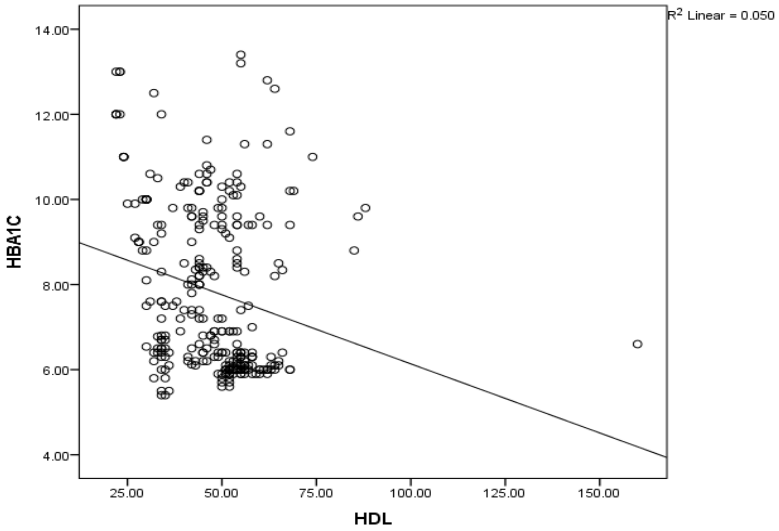


Fig. 8: Correlations of HbA1c with HDL.

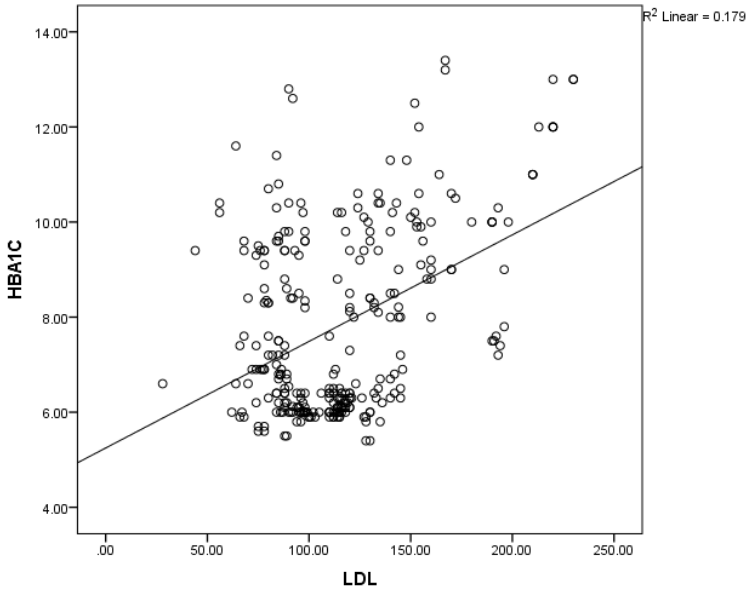


Fig. 9: Correlations of HbA1c with LDL.

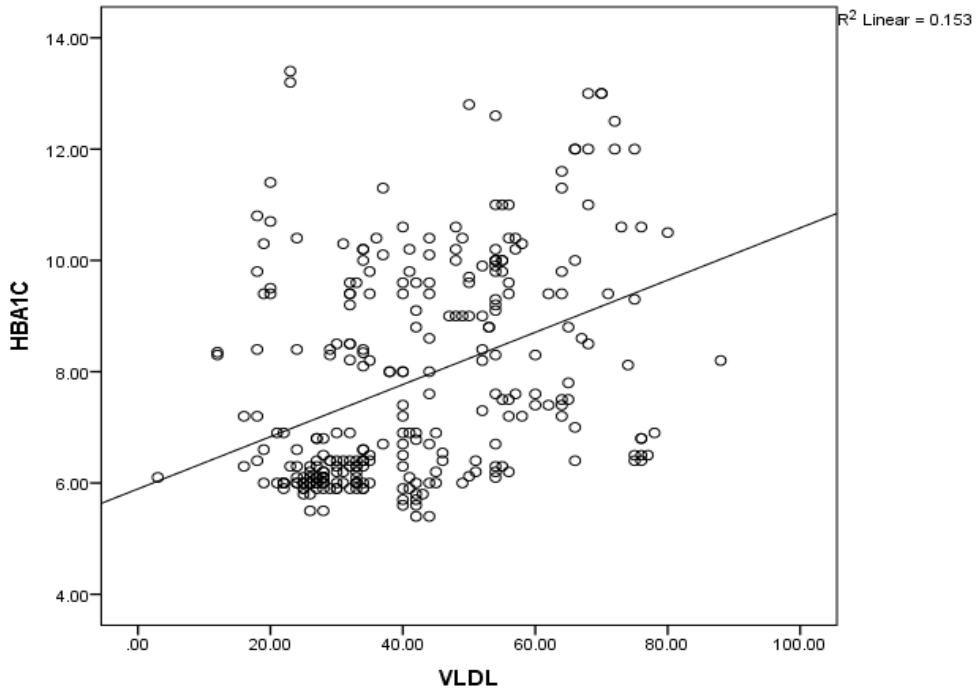


Fig. 10: Correlations of HbA1c with VLDL.

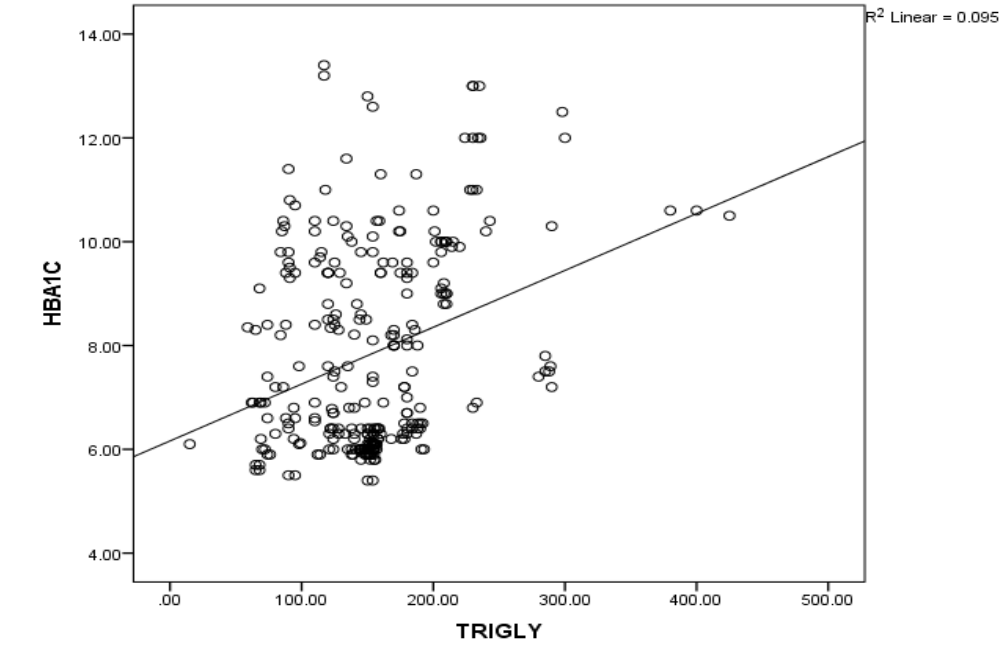


Fig. 11: Correlations of HbA1c with TRIGLYCERIDE.

The HbA1c also correlated with the BMI with a Pearson's correlation co-efficient of 0.474.

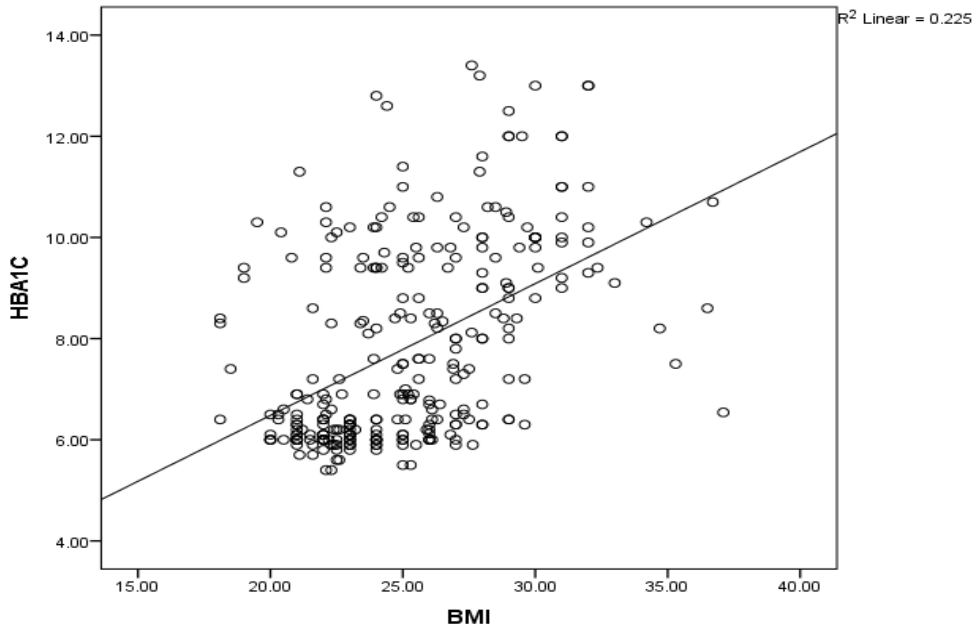


Fig. 12: Correlations of HbA1c with BMI.

When we did regression analysis for the same data with HbA1c as the dependent variable, we observed that the HbA1c was dependent on the following variables: FBS, PPBS, LDL, VLDL and BMI with statistical significance. The beta value suggests the amount with which the dependent variable changes with every one unit change in the variable. So for one unit increase in FBS and PPBS, HbA1c will increase by 0.408 and 0.201 respectively. The values of the beta and the t-values are depicted in the table below.

Table-8: Correlation of HbA1c and various variables and dependence of HbA1c on variables.

Coefficients							
	Unstandardized Coefficients		Standardized Coefficients	t-value	p-value	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	-.275	.690		-.399	.690	-1.633	1.083
FBS	.013	.003	.408	4.731	.0001**	.008	.019
PPBS	.005	.002	.201	2.157	.032*	.000	.009
TC	-.001	.002	-.042	-.614	.540	-.006	.003
HDL	.006	.006	.039	.916	.361	-.007	.018
LDL	.015	.003	.286	4.618	.0001**	.009	.022
VLDL	.014	.006	.116	2.504	.013*	.003	.025
TRIGLY	-.001	.002	-.022	-.387	.699	-.005	.003
UREA	.024	.007	.173	3.200	.002**	.009	.038
CREAT	-.299	.247	-.065	-1.214	.226	-.785	.186
BMI	.101	.022	.183	4.487	.0001**	.057	.145

To summarize our findings we have tabulated the mean values between the two groups.

Table- 9: Statical analysis of various parameters among cases and control group.

	GROUP						t-value	p-value
	Control			Cases				
	N	Mean	SD	N	Mean	SD		
AGE	145	53.61	10.86	145	51.06	10.32	2.045	.042*
BMI	145	23.57	2.54	145	26.86	3.66	8.907	.0001**
FBS	145	112.82	36.97	145	176.66	60.04	10.903	.0001**
PPBS	145	158.67	62.16	145	264.11	68.63	13.712	.0001**
TC	145	161.76	30.40	145	216.24	61.92	9.510	.0001**
HDL	145	50.52	13.00	145	44.44	13.20	3.950	.0001**
LDL	145	103.84	20.37	145	127.54	44.97	5.779	.0001**
VLDL	145	35.16	13.60	145	47.86	16.26	7.217	.0001**
TRIGLY	145	138.76	36.47	145	167.94	65.69	4.677	.0001**

DISCUSSION

Demographic profile

The whole population that was being studied had a mean age of 52.33 years. The mean age of the people in the control group was 53.61 ± 10.86 years, whereas the mean age of the people in the case group was 51.06 ± 10.32 years. Due to the fact that diabetes affects individuals of various ages, the age group that is being researched can shift based on the demographics of the patients who took part in the study. When we look at research that involves the diabetes population, we discover that the mean age was 52.6 ± 10.6 in a study by Schamarek et al that included 513 patients.[77] This information is found when we examine studies that include research that involves the diabetic population. Patients with diabetes who participated in the study that Pradeepa and her colleagues carried out were, on average, 54.7 ± 11.1 years.[27] A research conducted by Jung et al. discovered that the average age of patients who had been diagnosed with autonomic neuropathy was 52.4 years old ± 10.0 years.[78] These findings are consistent with the findings of our analysis. Eeg Olofsson et al split the participants of their research into groups based on their HbA1c levels. Those with the highest HbA1c values had a mean age of 62 that was only a little lower than the others.[65] In their study of 206 diabetic patients, Alzahrani and colleagues reported that the mean age was 64.66 ± 10.99.[76]

In the course of our investigation, we found that the number of women to be somewhat higher than the number of males. The findings of the study that Pradeepa and her colleagues conducted both point to the same conclusion.[27] In the studies that Schmarek, Jung, Alzahrani, and Ozder carried out, the proportion of male to female participants was reversed. These studies contained a larger number of male participants.[70,76-78]

Biochemical Profile

The mean HbA1c for the control group was calculated at 6.21 ± 0.34, whereas the mean HbA1c for the cases was calculated at 9.47 ± 1.47. Although Obata et al. reported mean HbA1c readings of 8.9, Jung's investigation discovered that the mean was only 7.9.[78,79] Jorsal et al. found a mean HbA1c of 9.4, although Snehalatha et al. and Schamarek et al. discovered mean readings that were lower than other studies (6.3 and 6.5 respectively).[77,80,81] As a consequence of this, we are able to arrive at the understanding that the mean HbA1c is not stable and that it changes depending on the population that is being studied.

Both fasting blood sugar and postprandial blood sugar readings are substantially different between the two groups, with higher values being found in cases than in controls for both measures. These parameters' mean values, which Jung, Schmarek, Pradeepa, Hansen, and Obata et al. discovered via their study, were the comparable as the mean values that we discovered through our own investigation.[22,77-79,82]

Lipid profile

In the course of our research, a comprehensive lipid profile was analyzed. The mean levels of total cholesterol, LDL cholesterol, very low density lipoprotein cholesterol, and triglycerides in our patients were 216.24, 127.54, 47.86, and 167.94 respectively. This was a substantial increase when compared to the mean that was shown in the control group (161.76, 103.84, 35.16 and138.76 respectively). When compared to the HDL levels seen in the control group, which were much higher, the cases had HDL levels of 44.4. The results of our research were consistent with those found in Alzahrani's previous research.[76] They found that the mean levels of total cholesterol, LDL cholesterol, and triglycerides were respectively 170.24, 143.65, and 103.78. According to their research, the HDL was 47.56. They separated their participants into two groups, just as we did: those with excellent control and those with poor control, depending on whether their HbA1c was less than 7 or greater than 7. With the exception of TGs (p=0.020) and HbA1c (p0.001), they could not find any significant differences between the two groups in any of the parameters they tested. According to the findings of the research conducted by Ozder et al., the mean values for total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol in patients were as follows: 227.6 57.7 mg/dl, 221.6 101.1 mg/dl, 31.5 6.7 mg/dl, and 136.5 43.7 mg/dl, respectively. Male patients had mean values of 219.1 34.7 mg/dl for total cholesterol, 250.0 100.7 mg/dl for total fat, 30.2 7.4 mg/dl for high density lipoprotein, and 125.7 21.4 mg/dl for low density lipoprotein, respectively. Patients who had diabetes were shown to have considerably lower mean blood levels of HDL and significantly higher mean serum levels of total cholesterol, triglycerides, and low-density lipoprotein (LDL) (p 0.001). The cross-sectional research that was conducted by Pandey et al. included a total of 120 individuals who were diagnosed with type 2 diabetes mellitus. They split the patients into groups according to their glycemic control and found that patients who had poor glycemic control had higher mean total cholesterol and mean high density lipoprotein levels than patients who had superior glycemic control. This was seen in both groups of patients. Patients who had poor

control of their glucose levels had greater mean levels of triglycerides and low density lipoprotein than patients who had tight control of their glucose levels.[83] An observational cross-sectional research was conducted by Panjeta et al. and included 60 diabetes patients. Patients were divided into two groups based on their HbA1c values: Group 1: HbA1c 7% (good glycemic control), and Group 2: HbA1c > 7%. (poor glycemic control). The group of patients who had excellent glycemic control had median serum triglyceride concentrations of 1.48 (1.15–2.22) mmol/L, whereas the group of patients who had poor glycemic control had median serum triglyceride concentrations of 2.15 (1.4–3.32) mmol/L. Those who had excellent control of their blood glucose levels had considerably lower median serum triglyceride concentrations compared to patients who had poor control of their blood glucose levels. The group of patients who had excellent glycemic control had a ratio of TAG to HDL cholesterol that was considerably lower than the value obtained in the other group. ($p = 0.025$) The ratio of TAG to HDL cholesterol had a median value of 1.75 0.19 in a group of patients who had excellent glycemic control, whereas the median value of the ratio was 2.79 0.42 in a group of patients who had poor glycemic control.[72] According to the research carried out by Hussain et al., the mean and standard deviation for total cholesterol, total fat, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol were 206, 256, and 40.85, respectively.[21] The research conducted by Sirsikar et al comprised a total of 150 participants: 75 patients diagnosed with type 2 diabetes and 75 gender-matched control individuals who did not have diabetes and seemed to be in good health. The total cholesterol, total fat, low-density lipoprotein (LDL-C), and very low-density lipoprotein (VLDL) levels were considerably higher in the cases.[84]

Correlation of HbA1c with other variables

When we investigated the correlations of HbA1c with our key variables, we found that the link with blood sugar levels, lipid profile, and BMI was statistically significant. Both the fasting plasma glucose level (FBS) and the postprandial blood sugar level (PPBS) were shown to have an association with HbA1c, with a Pearson's correlation coefficient of 0.645 and 0.704, respectively. There was a strong association between total cholesterol, LDL, VLDL, and triglycerides and HbA1c, as shown by the Pearson's correlation co-efficient values, which were correspondingly 0.542, 0.423, 0.391, and 0.309. The value of the Pearson correlation coefficient between HDL and HbA1c was -0.242, indicating that there was an inverse association between

the two variables. When we compare our findings to those of other research, we find that there is a consistent pattern. A cross-sectional research was carried out by Baranwal et al., and it included 168 patients who came in succession to the endocrinology outpatient clinic. All of the patients had either been diagnosed with type 2 diabetes or were already receiving therapy for it. It was discovered that HbA1c had significant positive correlation with total cholesterol (r = 0.257, p 0.01), low-density lipoprotein (r = 0.785, p 0.01), and total cholesterol (r = 0.272, p 0.01); significant negative correlation with high-density lipoprotein (r = -0.897, p 0.01); and significant positive correlation with HDL/LDL ratio (r = -0.690, p 0.01). In order to appreciate the value of HbA1c as a marker of individuals with dyslipidemia were split into two groups: those with excellent glycemic control and those with poor glycemic control, categories determined by the levels of HbA1c, with <7% and >7% being the appropriate cutoffs for each group. It was discovered that individuals who had excellent glycemic control had reduced mean values for total cholesterol, low-density lipoprotein cholesterol, and total cholesterol than individuals who had poor control of their glucose levels. However, the mean value of HDL and the ratio of HDL to LDL was shown to be greater in individuals who had excellent control of their blood sugar compared to those who had poor control of their glycemic status. At the threshold of p less than 0.05, these differences were statistically significant.[15]

According to the findings of Mohammed and colleagues, the level of glycated haemoglobin, may be used as a direct predictor of elevated TG and contributes to the indirect estimation of the risk of macro- and microvascular issues. HbA1c also showed direct and significant correlations with cholesterol with r value 0.6445, TG with r value 0.5426, LDL-C with r value of 0.3584, VLDL r value 0.2245, and L/H R value of 0.3416 with a strong positive correlation; however, HDL-C showed a negative correlation with r value.[69] These findings were discovered by Sirsikar et al in their study of 150 subjects. They came to the conclusion that there were substantial associations between HbA1c and all of these variables. A linear association between HbA1c and dyslipidemia as well as lipid markers suggest towards the efficacy of HbA1c for screening high-risk diabetes patients.[84] The Pearson correlation coefficient (r) was used in the research carried out by Tagore and colleagues to determine whether or not there was a connection between HbA1c and the different lipid profile characteristics. There was a link between HbA1c and total cholesterol (r = 0.557; p 0.0001) and low-density lipoprotein (r = 0.592; p 0.001 accordingly), and this

association was shown to be very significant. It was shown that there is a connection between HbA1c and TGs, and their correlation was 0.517; however, this did not approach the level of statistical significance required to be considered significant. On the other hand, a strong negative correlation was shown to exist between HDL and HbA1c (r = -0.449; p 0.001).(85) In individuals with type 2 diabetes, Hussain and colleagues investigated whether or not there was an association between HbA1c and serum lipid profile. They analyzed 401 patients and discovered that there was a substantial positive association between HbA1c and TC (r = 0.257, p = 0.000). This link was determined to be statistically significant. A significant connection was also found between HbA1c and TG (r = 0.235, p = 0.000). A statistically insignificant negative association between HbA1c and HDL-C was established (r = -0.024, p = 0.625). This connection was shown to exist between HbA1c and HDL-C. In addition, it was discovered that HbA1c had a positive and statistically significant relationship with LDL-C (r = 0.153, p = 0.002) as well as the ratio of LDL-C to HDL-C (r = 0.150, p = 0.003). A linear regression analysis revealed that HbA1c was a predictor of hypercholesterolemia (p = 0.000, R2 = 0.078), LDL-C (p = 0.002, R2 = 0.002), and TG (p = 0.000, R2 = 0.055). In addition to glycemic management, they came to the conclusion that HbA1c may also be used as a possible biomarker for predicting dyslipidemia in individuals who have type 2 diabetes.[21] Additionally, Panjeta and colleagues discovered a statistically significant positive association between the levels of glycated haemoglobin and the blood concentrations of triglycerides (r = 0.375), as well as a strong positive connection between the level of glycated haemoglobin and the ratio of TAG to HDL-c.[72] According to the findings of the research carried out by Pandey and colleagues, HbA1c has a statistically significant link with total cholesterol, triglycerides, and LDL, as well as an inverse correlation with HDL.[83] We also came across a few research, such as the ones by Alzahrani et al and Samdani et al, that also sought to check for the correlation, but their findings failed to achieve statistically significant values.[76,86]

Based on the aforementioned findings, we may infer that HbA1c correlates substantially with the lipid profile. This link is of major importance since HbA1c is often the first test performed on newly diagnosed diabetics and also the most frequently repeated test used to check glycemic management. HbA1c may serve as a dual marker for glycemic state and dyslipidemia, enabling early detection of the abnormal lipid profile. In turn, this would result in early intervention for

the treatment of dyslipidemia, hence reducing the risk of stroke and myocardial infarction. There is a need for large, longitudinal, and multicentric research to corroborate these results, since this association offers significant potential.

CONCLUSIONS

1. Diabetes Mellitus is a rapidly expanding epidemic that causes macrovascular and microvascular problems, imposes a heavy financial and emotional toll on both sufferers and the healthcare system, and results in an excess of morbidity and death.
2. The mean HbA1c for the control group was calculated at 6.21 $\pm$ 0.34, whereas the mean HbA1c for the cases was calculated at 9.47 $\pm$ 1.47. The difference as expected was statistically significant.
3. The lipid profile was also significantly different between the two groups. While the cholesterol, triglyglycerides, LDL and VLDL was higher in cases than controls, HDL was lower in cases. The same has been concluded in various studies on similar topic.
4. HbA1c significantly correlated with the fasting and postprandial blood glucose levels.
5. HbA1c showed a statistically significant positive correlation with the total cholesterol levels and triglycerides. The correlation was in concordance with the findings from the previous literature.
6. There was a significant correlation between HbA1c and lipoprotein levels, namely LDL and VLDL. We observed as the HbA1c rose, so did the LDL and VLDL levels.
7. HbA1c showed a negative correlation with HDL levels. The correlation was statistically significant and in line with previous findings from other studies.
8. It is reasonable to conclude that HbA1c has a strong association with lipid levels. Since HbA1c is often the first test conducted on newly diagnosed diabetics and the most commonly repeated test used to evaluate glycemic control, this connection is of crucial relevance.
9. Dyslipidemia may be detected early on if HbA1c is used as a marker for both the diabetic status and the aberrant lipid profile. As a consequence, the risk of cardiovascular diseases including stroke and myocardial infarction might be lowered with earlier diagnosis and treatment of dyslipidemia.
10. Given the promising nature of the observed correlation, more large-scale, longitudinal, and multicentric studies are warranted to confirm these findings.

SUMMARY

A rapidly spreading epidemic, diabetes mellitus causes both macrovascular and microvascular issues, places a significant financial and emotional burden on patients and the healthcare system, and increases morbidity and mortality rates. While the mean HbA1c for the patients was determined at 9.47 + 1.47, the mean HbA1c for the control group was 6.21 + 0.34. As predicted, the difference was statistically significant. Between the two groups, there were also noticeable differences in the lipid profiles. While the HDL was lower in cases compared to controls, the cholesterol, triglycerides, LDL, and VLDL were all greater in cases. The same conclusion has been reached in numerous investigations on a related subject. Significant correlations were found between HbA1c and postprandial and fasting blood glucose levels. The total cholesterol and triglyceride levels were positively correlated with HbA1c in a statistically significant way. The association matched the conclusions from the earlier literature. HbA1c levels and lipoprotein concentrations, specifically LDL and VLDL, were significantly correlated. We saw that the levels of LDL and VLDL increased as the HbA1c increased. HDL levels and HbA1c revealed a negative connection. The correlation held up to prior findings from other studies and was statistically significant. It makes sense to draw the conclusion that HbA1c and lipid levels are strongly correlated. This link is extremely important because HbA1c is frequently the first test performed on newly diagnosed diabetics and the most frequently repeated test used to assess glycemic management. If HbA1c is utilised as a diagnostic for both the presence of diabetes and the abnormal lipid profile, dyslipidemia may be identified early. Consequently, earlier detection and treatment of dyslipidemia may reduce the risk of cardiovascular illnesses such as stroke and myocardial infarction. It is necessary to conduct more large-scale, longitudinal, and multicentric research to corroborate these results because of the observed correlation's promising nature.

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ANNEXURE-1

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Document Information

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ANNEXURE-1

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

F. No. SU/2021/1830[17] Dated: 17.07.2021

Subject: Grant of Ethical Clearance for Thesis Protocol Topic **"ASSOSIATION BETWEEN HBA1C AND LIPID PROFILE IN PATIENTS OF T2DM",** submitted by **Dr. Nitin Rana,** MD/MS student of Batch 2020-2021, Department of **General Medicine**.

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"ASSOSIATION BETWEEN HBA1C AND LIPID PROFILE IN PATIENTS OF T2DM", Dr. Nitin Rana** is informed that the Thesis Protocol submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 19.03.2021, 22.03.2021, 24.03.2021, 25.03.2021, 26.03.2021, 27.03.2021 and 06.04.2021. The recommendations of the **Screening Committee** were considered in detail by the Institutional **Ethics Committee** in its meeting held on **12.07.2021** and the same was **approved** by the **Ethics Committee**.

He /She is informed accordingly for further necessary action.

[Dr. V. P. Gupta]
REGISTRAR

SANTOSH "DEEMED TO BE UNIVERSITY" REGISTRAR GHAZIABAD, NCR DELHI

Dr. Nitin Rana
MD/MS student of batch 2020-2021
Department of General Medicine
Email: dr.nitinrana1111@gmail.com

Copy to:

1. The Vice Chancellor
2. The Dean, SMC&H
3. The Dean – Research
4. The Dean – Academics
5. The Director - IQAC
6. Dr. Shivani Bansal (Guide)
7. HOD of **General Medicine**

ANNEXURE-2

CLINICAL PROFORMA

Particulars of the patient:

1. Name:
2. Age:
3. Sex:
4. Occupation:
5. D.O.A
6. D.O.D

History:

Presenting complaints (with duration, onset, associated factors)

1. Polyuria
2. Polyphagia
3. Polydypsia
4. Weight loss/gain
5. Nocturnal diarrhoea/constipation
6. Hypoglycaemic unawareness
7. Bladder/bowel incontinence
8. Impotence
9. Postural dizziness
10. History of significant cardiovascular / cerebrovascular disease / thyroid disorder.

Personal History:

1. Dietary-
2. Sleep-
3. Bladder and bowel-
4. Smoking-
5. Alcohol-
6. Treatment History:

General Physical examination:

1. Built
2. Nutrition
3. Height(cm)/Weight(cm)/Waist(cm)
4. BMI
5. Pallor/Icterus/Clubbing/Cyanosis/Lymphadenopathy/Oedema
6. Vitals: BP
7. Pulse

Investigations:

1. Fasting Blood glucose
2. Post Prandial Blood Glucose
3. Blood Urea
4. Serum Creatinine
5. Urine routine, microscopic analysis and microalbumin
6. Lipid profile
7. HbA1c

CBC	LFT	KFT	LIPID PROFILE
Hb		Urea	TG
TLC		Creatinine	HDL , LDL
DLC		Sodium	VLDL
Platelet		Potassium	Total cholesterol
Hct	T.Protein	HbA1c	FBS
	Albumin	Urine-R/M, microalbumin	PPBS

ANNEXURE-3

CONSENT FORM

SANTOSH MEDICAL COLLEGE AND HOSPITAL, GHAZIABAD, U.P.

I _____S/o/D/o/W/o_____R/o_____ have been explained about the risks of this study by Dr. Nitin Rana. I volunteer to take part in the study titled **"ASSOCIATION BETWEEN GLYCATED HEMOGLOBIN (HbA1c) AND LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS"** and have also been explained the purpose of this study to my satisfaction. I have been informed that in case of any adverse reaction, the treatment and compensation will be the responsibility of the investigator.

My details will be kept confidential and I have the freedom to withdraw from the study at any point of time. I have been explained that I am under no risk while participating in this study.

I hereby give my consent to be enrolled in this study.

Signature of volunteer/guardian Signature of investigator

Telephone number

Signature of witness Date:

सहमति पत्र

संतोष मेडिकल कॉलेज और अस्पताल, गाजियाबाद, यू.पी.

मुझे _____S/o/D/o/W/o_____R/o_____ को डॉ. नितिन राणा द्वारा इस अध्ययन के जोखिमों के बारे में समझाया गया है। मैं स्वेच्छा से **"ASSOCIATION BETWEEN GLYCATED HEMOGLOBIN (HbA1c) AND LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS"** शीर्षक वाले अध्ययन में भाग लेना चाहता हूं। और मेरी संतुष्टि के लिए इस अध्ययन के उद्देश्य के बारे में भी बताया गया है। मुझे सूचित किया गया है कि किसी भी प्रतिकूल प्रतिक्रिया के मामले में, उपचार और मुआवजे की जिम्मेदारी अन्वेषक की होगी।

मेरा विवरण गोपनीय रखा जाएगा और मुझे किसी भी समय अध्ययन से हटने की स्वतंत्रता है। मुझे समझाया गया है कि इस अध्ययन में भाग लेने के दौरान मुझे कोई जोखिम नहीं है।

मैं इस अध्ययन में नामांकित होने के लिए अपनी सहमति देता हूं।

स्वयंसेवी/अभिभावक के हस्ताक्षर

अन्वेषक के हस्ताक्षर

टेलीफोन नंबर

गवाह के हस्ताक्षर दिनांक:

PATIENT INFORMATION SHEET

1. OPD no:
2. Name:
3. Age:
4. Sex:
5. Address:
6. Phone number:

We invite you / your patient to participate in the research study titled study of being conducted in Santosh Medical College and Hospitals, Ghaziabad.

In accordance with the rules of ethical committee, a written consent will be taken. The relevant history will be asked and clinical examination of the patient will be done. Only the results of the investigations that you / your patient has already underwent as standard of care for such condition, are required for the study and no additional investigation will be done for the study. Special investigations, if any, will be sent as per the discretion of the treating physician and no additional investigation will be conducted for the purpose of the study You / your patient identity will be kept confidential throughout the study and thereafter. You are free to withdraw from the study any time without any loss of rights to undergo treatment in this hospital.

For any further questions regarding the study you can contact the investigator of study at the following number: Dr. Nitin Rana [+91 7206455757].

रोगी सूचना पत्र

1. ओपीडी संख्या:

2. नाम:

3. आयु:

4. सेक्स:

5. पता:

6. फोन नंबर:

हम आपको/आपके रोगी को संतोष मेडिकल कॉलेज और अस्पताल, गाजियाबाद में किए जा रहे अध्ययन शीर्षक के शोध अध्ययन में भाग लेने के लिए आमंत्रित करते हैं।

नैतिक समिति के नियमों के अनुसार लिखित सहमति ली जाएगी। प्रासंगिक इतिहास पूछा जाएगा और रोगी की नैदानिक परीक्षा की जाएगी। अध्ययन के लिए केवल उन जांचों के परिणाम की आवश्यकता होती है जिन्हें आप/आपके रोगी ने ऐसी स्थिति के लिए देखभाल के मानक के रूप में पहले ही झेला है और अध्ययन के लिए कोई अतिरिक्त जांच नहीं की जाएगी। विशेष जांच, यदि कोई हो, उपचार करने वाले चिकित्सक के विवेक के अनुसार भेजी जाएगी और अध्ययन के उद्देश्य के लिए कोई अतिरिक्त जांच नहीं की जाएगी आप/आपके रोगी की पहचान पूरे अध्ययन के दौरान और उसके बाद गोपनीय रखी जाएगी। आप इस अस्पताल में इलाज कराने के अधिकार के किसी भी नुकसान के बिना किसी भी समय अध्ययन से हटने के लिए स्वतंत्र हैं।

अध्ययन के संबंध में किसी और प्रश्न के लिए आप अध्ययन के अन्वेषक से निम्नलिखित नंबर पर संपर्क कर सकते हैं: डॉ. नितिन राणा [+91 7206455757].

MASTER CHAT

CASES

S.no	AGE	SEX	HBA1C	FBS	PPBS	TC	HDL	LDL	VLDL	TRIGLY	UREA	CREAT	BMI
1	62	MALE	8.3	146	262	160	45	132	29	128	37	1.06	18.1
2	62	MALE	8.4	146	264	160	45	130	29	125	37	1.06	18.1
3	30	MALE	7.4	180	215	124	55	66	40	74	30	0.98	18.5
4	48	FEMALE	9.4	180	227	170	57	127	32	129	25	0.8	19
5	48	FEMALE	9.2	182	221	170	51	125	32	134	24	0.82	19
6	60	MALE	10.3	220	321	290	39	193	58	290	71	2.84	19.5
7	54	FEMALE	10.1	210	314	214	54	150	44	154	22	0.84	20.4
8	60	FEMALE	9.6	184	289	130	45	98	56	110	24	0.62	20.8
9	32	FEMALE	11.3	242	427	248	62	148	37	187	44	1.02	21.1
10	67	MALE	8.6	89	178	145	44	89	67	126	23	1.2	21.6
11	65	FEMALE	7.2	140	188	156	34	82	64	130	25	1.15	21.6
12	45	FEMALE	10.6	184	319	210	54	134	48	174	34	0.82	22.1
13	45	MALE	10.3	208	315	170	55	124	31	134	24	0.85	22.1
14	55	MALE	9.4	280	321	180	50	68	32	180	36	1.11	22.1
15	55	MALE	9.6	282	344	180	50	68	32	180	36	1.11	22.1
16	45	MALE	10	200	314	170	50	129	34	138	21	0.78	22.3
17	65	FEMALE	8.3	161	312	168	34	78	54	186	33	0.92	22.3
18	45	MALE	10.1	203	312	170	53	127	37	135	23	0.9	22.5
19	72	FEMALE	7.2	88	146	160	45	88	56	178	40	1.2	22.6
20	38	MALE	10.2	258	314	210	69	116	57	175	56	2	23
21	68	MALE	9.4	180	233	220	68	120	62	160	25	1.16	23.4
22	55	MALE	8.3	210	196	140	47	80	12	65	57	2.25	23.4
23	55	FEMALE	9.6	180	240	240	60	130	42	162	32	0.88	23.5
24	55	MALE	8.35	210	198	140	43	79	12	59	52	2.1	23.5
25	70	MALE	8.1	190	398	210	30	134	34	154	34	0.92	23.7
26	55	MALE	7.6	144	188	130	34	110	44	120	24	0.6	23.9
27	65	MALE	9.4	186	269	161	33	93	35	175	28	0.85	23.9
28	37	FEMALE	10.2	196	256	144	44	56	34	110	22	0.54	23.9
29	38	FEMALE	10.2	210	330	240	44	152	41	201	33	0.98	24
30	55	FEMALE	9.4	188	242	240	62	134	44	160	34	0.98	24
31	60	MALE	8.2	123	244	180	48	98	35	84	20	0.65	24
32	47	FEMALE	12.8	196	234	168	62	90	50	150	40	1.17	24
33	56	FEMALE	9.4	180	221	145	48	78	19	95	45	0.88	24
34	75	MALE	9.4	183	169	156	54	78	64	120	33	1.03	24.2
35	49	FEMALE	10.4	255	313	160	54	56	24	110	24	0.84	24.2
36	52	MALE	9.7	188	242	150	45	85	50	114	30	0.74	24.3
37	47	FEMALE	12.6	186	234	168	64	92	54	154	44	1.17	24.4
38	38	FEMALE	10.6	218	312	240	46	154	40	200	34	0.98	24.5
39	35	FEMALE	8.4	249	376	150	44	92	52	110	32	0.72	24.7
40	52	MALE	7.4	165	221	130	42	88	62	124	18	0.84	24.8
41	54	MALE	8.5	180	298	148	54	120	68	124	44	0.86	24.9
42	52	FEMALE	7.5	78	202	288	37	191	56	288	69	2.7	25
43	70	FEMALE	11	313	413	280	74	164	54	118	55	1.24	25
44	45	MALE	9.6	113	225	231	42	156	33	169	29	0.84	25
45	50	FEMALE	8.8	288	361	240	85	114	65	142	24	1	25
46	58	FEMALE	7.5	169	188	180	57	85	64	184	38	1.1	25
47	54	FEMALE	11.4	225	411	210	46	84	20	90	24	1	25
48	56	FEMALE	9.5	182	244	145	45	75	20	91	46	0.84	25
49	58	FEMALE	7	166	191	180	58	84	66	180	37	1.1	25.1
50	38	FEMALE	9.4	147	242	180	44	44	20	88	27	0.99	25.2
51	62	FEMALE	8.4	217	311	200	54	70	24	74	48	1.16	25.3
52	38	MALE	10.4	130	172	240	52	90	44	124	36	0.92	25.4
53	50	FEMALE	9.8	299	366	240	88	118	64	145	26	1.1	25.5
54	52	FEMALE	7.2	78	214	290	39	193	58	290	71	2.84	25.6
55	52	FEMALE	7.6	84	210	287	38	192	57	289	70	2.8	25.6
56	62	FEMALE	10.4	110	188	200	41	135	57	159	20	0.71	25.6
57	38	MALE	8.8	130	178	240	54	88	42	120	34	0.9	25.6
58	58	FEMALE	9.6	205	283	220	86	84	40	200	38	0.88	25.6
59	52	MALE	7.6	140	219	160	34	68	54	98	19	0.87	25.6
60	47	FEMALE	8.5	144	249	160	65	95	32	120	24	0.81	26
61	45	MALE	7.6	78	214	156	31	80	60	135	21	1.1	26
62	86	MALE	8.3	129	214	180	56	80	60	170	36	1	26.2
63	50	FEMALE	8.21	142	223	160	44	144	32	140	39	0.84	26.3
64	50	FEMALE	8.5	142	232	160	40	140	30	144	35	0.84	26.3
65	45	MALE	9.8	245	321	155	49	88	18	90	24	0.92	26.3
66	45	MALE	10.8	349	501	155	46	85	18	91	27	0.95	26.3
67	47	FEMALE	8.34	140	256	160	66	98	34	122	25	0.82	26.5
68	42	MALE	9.4	184	246	138	34	88	56	120	34	0.92	26.7
69	45	FEMALE	9.8	166	233	180	50	98	35	84	26	0.9	26.8
70	45	MALE	7.5	78	212	156	30	85	65	125	28	1.2	26.9
71	69	FEMALE	7.4	154	248	210	44	74	64	154	34	0.84	26.9

S.no	AGE	SEX	HBA1C	FBS	PPBS	TC	HDL	LDL	VLDL	TRIGLY	UREA	CREAT	BMI
72	65	FEMALE	7.8	112	200	280	42	196	65	285	47	0.9	27
73	45	FEMALE	8	160	250	270	41	160	38	188	22	1	27
74	45	FEMALE	7.2	140	296	270	44	145	40	178	22	1	27
75	40	MALE	8	146	220	260	44	145	40	170	30	1.5	27
76	40	FEMALE	9.6	168	207	134	42	98	50	90	22	0.47	27
77	45	FEMALE	10.4	168	238	180	46	96	36	86	25	0.87	27
78	32	MALE	7.3	93	102	180	42	120	52	154	22	0.62	27.3
79	45	FEMALE	10.2	167	233	180	52	97	34	85	24	0.92	27.3
80	65	FEMALE	7.4	112	201	280	40	194	60	280	45	0.92	27.5
81	52	FEMALE	13.4	338	447	246	55	167	23	117	12	0.65	27.6
82	62	FEMALE	8.12	122	214	168	42	120	74	180	24	0.8	27.6
83	52	FEMALE	13.2	336	440	220	55	167	23	117	12	0.65	27.9
84	35	FEMALE	11.3	289	354	160	56	140	64	160	17	0.88	27.9
85	50	FEMALE	10	166	224	298	30	190	55	209	76	1.5	28
86	50	FEMALE	10	140	250	298	30	190	54	206	76	1.5	28
87	66	FEMALE	9	146	286	286	28	170	47	206	42	1.8	28
88	62	FEMALE	9	136	288	290	32	160	49	208	46	2.4	28
89	48	MALE	8	130	290	270	42	144	44	170	64	1.5	28
90	48	MALE	8	130	208	255	44	122	38	170	64	1.5	28
91	40	FEMALE	9.3	168	207	134	44	95	54	91	24	0.45	28
92	52	MALE	9.8	182	234	150	41	90	55	115	35	0.72	28
93	38	FEMALE	11.6	226	314	160	68	64	64	134	18	0.82	28
94	43	MALE	10.6	98	186	185	44	124	76	380	35	1.47	28.2
95	35	MALE	10.6	293	342	346	31	170	73	400	45	1.12	28.5
96	63	MALE	8.5	150	222	160	44	142	32	149	24	0.9	28.5
97	52	FEMALE	9.6	217	276	133	54	85	44	125	22	1	28.5
98	38	FEMALE	8.4	137	206	155	46	91	18	88	29	0.93	28.8
99	35	MALE	10.5	295	338	350	33	172	80	425	48	1.16	28.9
100	29	MALE	9.1	140	234	210	52	78	42	68	14	0.85	28.9
101	58	FEMALE	12	135	224	320	23	220	72	234	75	2	29
102	56	FEMALE	12	160	244	320	22	220	68	230	66	1.6	29
103	66	MALE	9	190	294	290	28	170	52	210	45	1.2	29
104	36	MALE	8.8	130	210	290	29	158	53	208	45	1.6	29
105	48	MALE	12.5	231	341	235	32	152	72	298	27	0.92	29
106	66	MALE	9	133	250	290	28	144	50	180	45	1.2	29
107	40	MALE	8	130	260	264	44	140	40	180	30	1.4	29
108	42	MALE	10.4	98	151	200	40	134	56	157	22	0.7	29
109	64	FEMALE	8.2	140	200	260	44	132	52	170	39	1.9	29
110	50	FEMALE	7.2	138	212	145	50	85	18	86	29	1.2	29
111	58	FEMALE	8.4	180	214	214	44	130	34	184	32	0.84	29.3
112	42	MALE	9.8	98	146	200	42	130	54	154	21	0.74	29.4
113	48	MALE	12	232	342	240	34	154	75	300	29	0.92	29.5
114	50	FEMALE	7.2	136	216	145	49	80	16	80	27	1.3	29.6
115	67	MALE	10.2	250	333	210	68	114	54	174	54	2.1	29.7
116	42	FEMALE	13	139	290	330	23	230	68	230	78	2.1	30
117	48	MALE	10	220	290	300	30	190	54	215	42	1.4	30
118	55	MALE	10	140	292	300	30	180	55	206	60	2	30
119	56	FEMALE	8.8	134	248	288	30	160	53	210	50	2	30
120	48	MALE	10	200	300	300	30	160	48	210	42	1.4	30
121	48	MALE	10	140	210	300	30	153	54	210	42	1.4	30
122	40	FEMALE	9.8	290	321	218	37	140	41	206	23	0.86	30
123	52	FEMALE	9.4	214	276	133	54	88	40	120	25	1.1	30.1
124	45	MALE	12	150	300	320	22	220	66	224	80	2.2	31
125	45	MALE	12	129	221	310	22	213	66	236	80	2.2	31
126	55	MALE	11	210	310	310	24	210	56	230	40	1.2	31
127	46	MALE	11	130	240	310	24	210	68	228	100	1.8	31
128	55	FEMALE	10	129	290	310	29	198	66	202	40	2.5	31
129	50	MALE	9	150	250	280	42	196	48	210	70	1.8	31
130	68	MALE	9.2	141	220	288	34	160	54	208	60	1.8	31
131	61	FEMALE	10.4	202	278	236	46	143	49	243	21	0.87	31
132	42	FEMALE	13	130	290	330	22	230	70	235	60	1.7	32
133	42	FEMALE	13	160	320	310	23	220	70	230	60	1.7	32
134	42	MALE	11	140	260	310	24	210	55	233	40	1.7	32
135	40	FEMALE	9.9	136	220	300	27	155	54	220	42	1.9	32
136	61	FEMALE	10.2	204	281	233	44	141	48	240	20	0.89	32
137	49	FEMALE	9.3	187	254	240	50	74	75	180	44	0.94	32
138	49	FEMALE	9.4	180	251	240	58	76	71	184	46	0.98	32.35
139	70	MALE	9.1	142	233	284	27	155	54	206	41	1.9	33
140	50	MALE	10.3	349	490	155	50	84	19	87	25	0.91	34.2
141	57	FEMALE	8.2	211	286	168	64	120	88	168	22	0.64	34.7
142	61	FEMALE	7.5	77	231	285	35	190	55	285	67	2.84	35.3
143	40	FEMALE	8.6	189	246	154	54	78	44	145	24	1.07	36.5
144	50	MALE	10.7	346	448	155	47	80	20	95	26	0.89	36.7
145	42	FEMALE	9.9	136	222	301	25	153	52	214	40	1.9	31

MASTER CHART

CONTROLS

S.no	AGE	SEX	HBA1C	FBS	PPBS	TC	HDL	LDL	VLDL	TRIGLY	UREA	CREAT	BMI
1	62	MALE	6.4	146	262	160	45	132	29	128	37	1.06	18.1
2	44	MALE	6.5	86	108	172	46	134	35	178	35	1.1	20
3	44	MALE	6	86	110	172	60	96	21	154	38	1.1	20
4	45	FEMALE	6.1	85	108	146	51	116	28	150	26	0.7	20
5	44	MALE	6	86	112	175	64	99	30	144	35	1.2	20
6	56	MALE	6	92	120	112	55	115	24	124	26	1.1	20
7	62	FEMALE	6.5	116	253	176	35	115	77	192	30	1.12	20.3
8	42	MALE	6.4	140	212	180	34	112	76	190	22	0.92	20.3
9	56	MALE	6	90	110	110	52	115	25	150	24	1.1	20.5
10	53	FEMALE	6.6	135	190	190	48	123	19	95	35	0.82	20.5
11	42	MALE	6.5	148	214	174	33	110	75	184	21	0.95	21
12	49	MALE	6.9	192	254	217	39	146	32	162	53	0.77	21
13	44	MALE	6.1	86	115	172	63	95	26	154	37	0.9	21
14	45	FEMALE	6.3	80	110	140	58	96	32	154	21	0.74	21
15	62	MALE	6	87	100	135	56	84	24	154	23	0.86	21
16	38	FEMALE	6.1	104	112	134	52	116	28	154	21	1.12	21
17	61	FEMALE	5.9	100	166	165	50	115	25	152	30	0.95	21
18	59	FEMALE	6.2	82	100	122	56	119	31	152	26	1	21
19	74	FEMALE	6	98	112	154	51	99	19	151	24	0.7	21
20	74	MALE	6.9	149	192	210	48	113	30	148	42	1.15	21
21	61	FEMALE	6	102	120	169	51	105	25	145	35	0.94	21
22	44	MALE	6	81	101	171	61	91	31	141	31	1.1	21
23	58	FEMALE	6.3	90	112	125	54	114	26	128	21	0.8	21
24	59	FEMALE	6.4	84	106	125	55	120	35	122	25	1.1	21
25	56	MALE	6	90	120	111	52	118	22	121	25	1.2	21
26	42	FEMALE	5.7	175	250	216	50	75	40	65	16	0.89	21.1
27	45	FEMALE	6.2	135	183	159	46	85	28	124	76	2.43	21.2
28	54	FEMALE	6.8	290	350	160	47	85	28	140	26	1.1	21.4
29	44	MALE	6.1	86	113	170	64	94	24	152	36	1.1	21.5
30	74	FEMALE	6	97	111	152	53	97	26	145	25	0.72	21.5
31	61	FEMALE	5.9	102	166	170	51	114	28	152	31	0.87	21.6
32	42	FEMALE	5.7	180	253	210	52	78	42	68	14	0.85	21.6
33	60	MALE	6.4	140	217	182	32	110	75	188	20	0.9	22
34	60	FEMALE	6.7	125	160	170	48	135	37	180	38	1.1	22
35	74	FEMALE	6.4	130	190	150	54	98	34	158	26	0.7	22
36	44	MALE	6	86	108	170	62	98	22	155	38	1.1	22
37	44	MALE	6.1	86	107	170	65	98	26	155	38	1.1	22
38	38	FEMALE	6.1	95	118	135	54	116	28	155	23	1.2	22
39	56	MALE	6	92	112	112	54	116	26	155	25	1	22
40	50	MALE	6.4	178	234	162	36	94	31	152	29	1.01	22
41	45	FEMALE	6.1	82	106	145	56	114	28	152	24	0.7	22
42	74	FEMALE	6	96	110	150	54	98	33	150	26	0.7	22
43	74	FEMALE	6	96	110	150	54	98	33	150	26	0.7	22
44	65	FEMALE	5.8	98	166	174	32	128	42	145	32	0.7	22
45	61	FEMALE	6	102	120	170	54	102	27	145	32	0.95	22
46	42	MALE	6	81	114	121	54	113	35	135	21	0.8	22
47	40	MALE	6.3	96	121	114	55	116	27	133	22	0.94	22
48	45	FEMALE	6.9	144	218	202	52	72	21	72	46	114	22
49	60	MALE	6.5	144	212	180	34	112	76	190	22	0.92	22.1
50	60	MALE	6.8	140	273	180	34	112	76	190	22	0.92	22.1
51	65	FEMALE	5.4	110	163	182	35	128	42	154	36	0.72	22.1
52	74	FEMALE	6	97	113	152	52	92	22	152	22	0.72	22.2
53	61	FEMALE	5.9	103	123	169	53	103	33	153	33	0.93	22.3
54	65	FEMALE	5.4	104	165	180	34	130	44	150	34	0.74	22.3
55	45	FEMALE	6.6	142	215	200	54	70	24	74	48	1.16	22.3
56	54	FEMALE	5.9	85	115	156	56	101	30	152	28	0.78	22.4
57	65	FEMALE	5.8	98	166	178	35	135	43	152	33	0.74	22.5
58	60	FEMALE	6	80	110	179	62	95	25	148	22	0.7	22.5
59	58	MALE	5.9	90	104	164	62	112	29	139	31	1.1	22.5
60	64	MALE	6.2	184	233	213	53	74	45	69	15	0.81	22.5
61	64	MALE	5.6	180	257	210	52	78	42	68	14	0.85	22.5
62	72	FEMALE	6.2	88	144	160	45	88	56	178	40	1.2	22.6
63	64	MALE	5.6	184	254	208	50	75	40	65	15	0.84	22.6
64	65	FEMALE	6.9	158	216	150	50	78	22	110	40	1.4	22.7
65	58	MALE	6.3	88	102	160	55	110	33	160	0.8	1	23
66	35	MALE	6.2	88	112	125	65	118	30	158	22	0.8	23
67	58	MALE	6.1	85	110	164	57	114	25	157	24	1	23
68	58	MALE	6.4	88	120	160	66	110	34	156	0.8	1	23
69	38	FEMALE	6.1	100	120	140	55	120	27	156	22	1.3	23
70	60	FEMALE	5.8	80	110	179	50	94	25	155	22	0.7	23

71	58	MALE	6.1	88	102	160	55	118	28	155	29	1	23
72	54	FEMALE	5.9	80	110	150	55	100	32	155	23	0.8	23
73	54	FEMALE	6	80	110	150	55	130	28	155	23	0.8	23
74	54	FEMALE	6	80	110	150	55	130	28	155	23	0.8	23
75	42	MALE	6.4	80	110	120	55	120	34	155	22	0.8	23
76	40	MALE	6.1	94	125	115	54	117	3	152	23	0.95	23
77	61	FEMALE	5.9	102	120	168	52	100	34	150	32	0.9	23
78	61	FEMALE	6.4	102	120	167	50	116	30	150	34	0.9	23
79	35	MALE	6.3	88	112	125	63	115	34	150	22	0.8	23
80	58	FEMALE	6.3	88	114	120	58	120	30	150	22	0.8	23
81	38	FEMALE	6.3	130	210	140	55	120	33	140	22	1.3	23
82	58	MALE	5.9	88	102	160	60	110	27	138	30	1	23
83	42	MALE	6	80	110	120	55	112	34	138	22	0.8	23
84	45	MALE	6.2	160	215	201	32	136	33	168	25	0.8	23.2
85	68	FEMALE	6.9	220	382	210	52	78	42	68	14	0.85	23.9
86	60	FEMALE	6.2	80	110	180	52	112	26	158	23	0.7	24
87	38	FEMALE	6.4	82	120	150	51	118	33	158	35	1	24
88	62	MALE	6	90	102	140	60	90	25	157	25	0.9	24
89	60	FEMALE	5.8	80	110	180	52	96	26	156	23	0.7	24
90	40	MALE	6.1	95	126	110	56	114	33	155	24	0.9	24
91	60	FEMALE	6	80	110	180	63	96	26	150	23	0.7	24
92	38	FEMALE	6	82	120	150	68	110	33	150	35	1	24
93	38	FEMALE	6	82	120	150	68	110	33	150	35	1	24
94	62	MALE	5.9	90	102	140	58	112	30	148	25	0.9	24
95	60	FEMALE	6.4	80	110	180	50	96	34	145	23	0.7	24
96	55	MALE	5.9	94	124	124	55	66	40	74	30	0.98	24
97	50	FEMALE	6.4	120	182	164	54	88	46	124	33	0.72	24.8
98	48	MALE	6.9	134	187	213	48	86.4	78	233	44	0.89	24.9
99	48	MALE	6.8	136	179	211	47	86	76	230	43	0.87	25
100	38	FEMALE	6.1	102	116	138	58	120	27	156	24	1.24	25
101	62	MALE	5.9	95	110	146	59	115	34	149	27	0.9	25
102	39	MALE	6	90	130	161	52	114	27	145	31	1.2	25
103	42	MALE	5.5	152	190	164	36	89	26	95	32	0.89	25
104	55	MALE	6	99	123	129	51	67	42	72	35	0.85	25
105	68	FEMALE	6.9	225	378	215	50	76	40	63	12	0.8	25
106	60	FEMALE	6.1	126	138	154	36	88	41	15	29	0.89	25
107	58	FEMALE	6.4	165	192	180	58	84	66	180	37	1.1	25.1
108	68	FEMALE	6.9	224	384	209	54	77	41	62	15	0.79	25.2
109	60	FEMALE	6.8	131	268	214	45	142	27	136	32	0.97	25.3
110	42	MALE	6.8	149	192	162	35	89	27	94	35	0.9	25.3
111	42	MALE	5.5	156	192	160	34	88	28	90	32	0.9	25.3
112	68	FEMALE	6.9	221	383	212	53	74	45	69	11	0.84	25.4
113	55	MALE	5.9	96	125	124	52	68	41	76	39	0.9	25.5
114	66	FEMALE	6.2	92	164	150	41	97	51	94	34	0.7	25.9
115	60	FEMALE	6	124	145	160	34	87	45	191	27	0.8	26
116	38	FEMALE	6.3	100	119	138	58	121	23	155	22	1.3	26
117	39	MALE	6	90	130	160	53	110	28	148	29	1.3	26
118	39	MALE	6.2	90	130	162	54	117	28	140	30	1.3	26
119	63	MALE	6.78	86	136	149	33	86	42	123	21	0.65	26
120	66	FEMALE	6.12	94	162	154	42	92	50	99	39	0.79	26
121	55	MALE	6	98	122	128	56	62	49	70	31	0.95	26
122	60	FEMALE	6	126	144	159	35	86	44	193	28	0.88	26.1
123	69	FEMALE	6.4	136	180	157	45	84	27	122	23	1.3	26.1
124	59	FEMALE	6.6	124	178	180	44	64	34	110	24	0.98	26.1
125	45	MALE	6.4	134	171	155	49	88	18	90	24	0.92	26.3
126	65	MALE	6.7	87	138	145	34	89	44	124	22	0.6	26.4
127	66	FEMALE	6.1	90	166	155	43	95	54	98	36	0.74	26.8
128	45	FEMALE	6.5	140	202	185	42	145	40	188	22	1	27
129	50	MALE	6.3	106	204	212	35	145	55	187	21	0.81	27
130	45	FEMALE	6.3	140	210	170	41	133	40	177	22	1	27
131	74	FEMALE	6	96	114	154	57	97	27	147	27	0.7	27
132	49	FEMALE	5.9	102	184	200	51	128	25	114	13	0.85	27
133	42	MALE	6.5	156	190	160	34	88	28	90	32	0.9	27.3
134	42	MALE	6.6	159	194	11.4	160	28	34	88	29	0.96	27.3
135	50	MALE	6.4	110	209	214	33	142	51	184	21	0.84	27.5
136	49	FEMALE	5.9	104	182	198	49	127	22	112	14	0.89	27.64
137	50	MALE	6.3	108	199	210	34	140	54	180	22	0.82	28
138	44	FEMALE	6.7	110	209	210	34	140	54	180	22	0.82	28
139	52	MALE	6.3	124	172	198	48	126	24	121	26	1.24	28
140	32	FEMALE	6.4	78	110	150	55	106	29	159	28	1.2	29
141	32	FEMALE	6.4	78	110	150	56	116	32	138	28	1.2	29
142	50	FEMALE	6.3	135	216	145	49	80	16	80	27	1.3	29.6
143	50	MALE	6.54	120	169	118	30	90	46	110	24	0.6	37.1
144	72	FEMALE	6.2	78	145	162	43	89	54	176	41	1.1	22.4
145	40	MALE	6.7	85	135	140	35	85	40	124	22	0.7	26